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MACMILLAN AND CO., LTD., LONDON.

A SYSTEM OF MEDICINE

First Edition, 1897
Second Edition, 1908

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PREFACE

It may perhaps be advisable to explain how it is that a volume of a *System of Medicine* contains a section devoted to a subject so special, and even surgical, as Diseases of the Ear. In the original edition Vol. IV. dealt with Diseases of the Liver, Pancreas, Kidneys, and Ductless Glands, and also contained articles on Diseases of the Nose and Throat, which thus introduced the Diseases of the Respiratory System in the fifth volume. In order to render the articles on the Nose and Throat more accessible to readers specially interested in these subjects, it appeared advisable in the second edition to issue them in a separate and smaller volume. When this had been decided, Sir Felix Semon, who has assisted the Editors with advice and help in the most generous manner, suggested the inclusion of the Diseases of the Ear, as this addition would probably be welcomed by many readers desirous of possessing a complete handbook of these subjects. This addition has been made under the supervision of Dr. M'Bride, who has constantly advised the Editors, and spared neither time nor trouble on the task.

The sections on Diseases of the Nose, Pharynx, and Larynx have been very considerably extended, and have been further supplemented by entirely new articles on Direct Laryngoscopy, Tracheoscopy, Oesophagoscopy, and Gastroscopy, by Mr. Waggett,

37481

and on Diseases of the Trachea, by Sir Felix Semon and Dr. Watson Williams. The addition of coloured Plates and of Figures in the text should add to the utility of the work, which, it is hoped, will serve as an authoritative treatise on these important subjects.

To Dr. A. J. Jex-Blake, who has compiled the Index, we are much indebted for a number of corrections.

CLIFFORD ALLBUTT.
H. D. ROLLESTON.

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In order to avoid frequent interruption of the text, the numbers indicative of items in the lists of "References" are only inserted in cases of emphasis, where two or more references to the same author are in the list, where an author is quoted from a work published under another name, or where an authoritative statement is made without mention of the author's name. In ordinary cases an author's name is sufficient indication of the corresponding item in the list.

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    - HYPERTROPHY OF THE PHARYNGEAL TONSIL.

I.—DISEASES OF THE NOSE

1. METHODS OF EXAMINATION OF THE NOSE

PROBABLY no kind of speculum examination needs so much practice as **rhinoscopy,** before it can be satisfactorily employed. The apparatus required is quite simple. Either direct sunlight, or good lamplight reflected from the forehead with the mirror used for laryngoscopy, is preferable. It is only in very exceptional cases that a more brilliant light is advantageous. A candle in a dark closet is better than even the lime-light in a well-lit room. Besides a good light there are needed a speculum, a few probes, an extra small laryngeal mirror, and a tongue-depressor. The form of speculum is not of much consequence, provided the blades are not fenestrated; otherwise the hairs in the vestibule obstruct the view. Thudichum's instrument, in three sizes, serves most purposes. Duplay's is probably the most comfortable for the patient, and at the same time has the advantage of forcibly thrusting the septum to one side when desired. The probe should not have a bulbous extremity, but should be roughened, so as to carry a mop of cotton-wool wrapped round the point for purposes of cleansing, or for the application of cocaine, adrenalin, and so forth. The best tongue-depressors are Fränkel's, made an inch longer than usual, or Türck's. A palate-hook is of little use: its insertion produces such an amount of sphincter-like contraction of the palate and constrictors of the pharynx, that it fails in the very cases in which it is most needed.

Anterior Rhinoscopy.—In the lower part of the fossa we observe the inferior turbinal, a fold of mucous membrane covering the inferior spongy bone and containing the plexiform arrangement of venous sinuses, which give the bodies their erectile property. This erectile tissue not only extends along the free border of the turbinal, being especially developed at the anterior and posterior extremities, but also as a thinner layer lines the floor and a portion of the septum. According to the degree of turgidity, we see to a greater or less distance along the inferior meatus; but frequently the swelling is so great that the channel is completely blocked. After exposure to cold air the inferior turbinal becomes swollen, and its usual bright red colour becomes darker. Yet, although this turgidity can hardly be considered pathological, it frequently comes under observation as a form of nasal obstruction, and as such may require treatment. Probing the inferior turbinal when in a state of turgescence, gives the impression of a sac loosely filled with fluid; indeed it fluctuates. But the application of a 5 per cent solution of cocaine, or of adrenalin solution, quickly induces collapse. According to the degree of prominence in the inferior turbinal, we see more or less of the free convex border of the middle turbinal, between which and the septum there is a clear passage, called the olfactory fissure. The latter varies considerably in width, even in health. Between the free margin of the middle turbinal, curved inwards on itself, and the outer wall of the fossa is a deep sulcus, corresponding with the concavity of the spongy bone, so that the free margin appears to hang downwards from the roof. It must be observed that between the anterior extremity of the middle spongy bone and the nasal process of the superior maxilla there is a free passage, which, however, is considerably narrower in the living subject than in the skeleton. In pathological conditions, more especially in oedematous rhinitis, it may be obstructed. The portion of the fossa below the upper margin of the inferior spongy bone is commonly spoken of as the inferior meatus; into it anteriorly opens the naso-lacrymal canal. Between the concavity of the middle turbinal and the convexity of the inferior is the middle meatus, into which the anterior ethmoidal cells open above and anteriorly.

On the outer wall, between two and three inches from the orifice of the ala, is found the semilunar hiatus lying between the upper margin of the unciform process and the projecting ethmoidal cell named by Zuckerkandl the *bulla ethmoidalis.* Upwards, this crescentic and valvular opening leads into the infundibulum, giving access to the frontal sinus; while running backwards, downwards, and somewhat outwards, curving under the bulla, it leads into the ostium maxillare. There are occasionally other accessory openings into the antrum; while it must be noted that the outer wall of the infundibulum is often separated from the cavity of the antrum by the two layers of mucous membrane alone. This point is of importance on account of the frequency with which frontal sinus suppuration is disguised by its pus finding entrance into the antrum.

The majority of individuals have more or less deflection of the septum, with greater or less development of certain bony ridges running from before backwards. This malformation often offers considerable difficulty in examining the side encroached upon. It is always associated with more or less thickening at the point where the curvature is sharpest. Sometimes there is thickening without deviation.

Posterior rhinoscopy is effected by inclining the laryngeal mirror upwards behind the soft palate. The difficulty consists in placing the mirror in position without touching the back of the tongue, the velum, or the posterior wall, but when these difficulties are circumvented, the structures brought into view are fairly easy of comprehension. For examination of the septum, turbinated bodies, and vault, the angle of the mirror should be made a right angle; while for the posterior naso-pharyngeal wall, the angle should be as wide as that of the laryngeal mirror. Fränkel's pharyngeal mirror, the inclination of which can be altered at will, is by some considered an advantage. It is almost always advisable to use a tongue-depressor as well. Firmness and gentleness in the use of the tongue-depressor are required for this sometimes exceedingly difficult manipulation. Relaxation of the soft palate may often be obtained by asking the patient to breathe through his nose, or to breathe in and out of his mouth as slowly and gently as possible. Yet the longer it takes, the more difficult does the examination become. Sometimes the application of a 5 per cent solution of cocaine may be advisable in conditions of great irritability; although only too often, in spite of producing anaesthesia, it aggravates the liability to reflex action. Holding the upper edge of the mirror just below and well behind the margin of the pendulous palate, a view of the post-nasal structures is obtained—a view, however, pieced together by a series of altered inclinations of the mirror. We see the two choanae separated by the vertical, in this situation very rarely deflected, septum. Into these project, downwards and inwards, the three turbinals with their intervening channels. From the prominence of what is called the uvula cushion, the inferior turbinals may be hidden. The middle meatus is not frequently encroached upon by hypertrophy or turgescence of the erectile tissue below it, while the superior meatus may appear to possess an unwarranted importance, seeing that it is only fully developed posteriorly. The spaces may be further narrowed by oedema of the mucous membrane on each side of the septum. This oedema often subsides after the application of cocaine or adrenalin.

Externally to the choanae we see the yellowish openings of the Eustachian tubes, with the salpingo-palatine folds in front, and the salpingo-pharyngeal folds behind. Externally to and behind this again is Rosenmüller's fossa, into which the Eustachian catheter sometimes inadvertently passes. At the highest point of the post-nasal cavity, running down the posterior wall, may be often seen the raised collection of lymphoid tissue known as the pharyngeal or Luschka's tonsil, in the centre of which a vertical depression may occasionally be seen. Running downwards on each side of this are frequently seen two parallel folds of mucous membrane, one on each side of the central raphe; but it is often impossible to say at what point this lymphoid tissue should be considered pathological. When it is largely hypertrophied it assumes the characteristic appearance of adenoids, and then interferes to a greater or less extent with a view of the upper part of the septum (*vide* Plate II.).

The normal colour of the nasal and naso-pharyngeal mucous membrane is not very easy to determine in cities where so many suffer from catarrh. In front the darkest portion is the inferior turbinal, the middle being much paler. Posteriorly the structures projecting into the choanae should present a pale grey colour, somewhat oedematous-

looking; while the mucous membrane of the naso-pharynx in general may be described as pinkish-grey, becoming darker as the pharynx is reached.
GREVILLE MACDONALD.

Transillumination. — Transillumination is a valuable method of examining the nasal accessory sinuses, and more particularly the antrum of Highmore; it is especially useful in assisting to determine the presence or absence of chronic suppuration in the antrum, although its use is by no means limited to this one morbid condition.

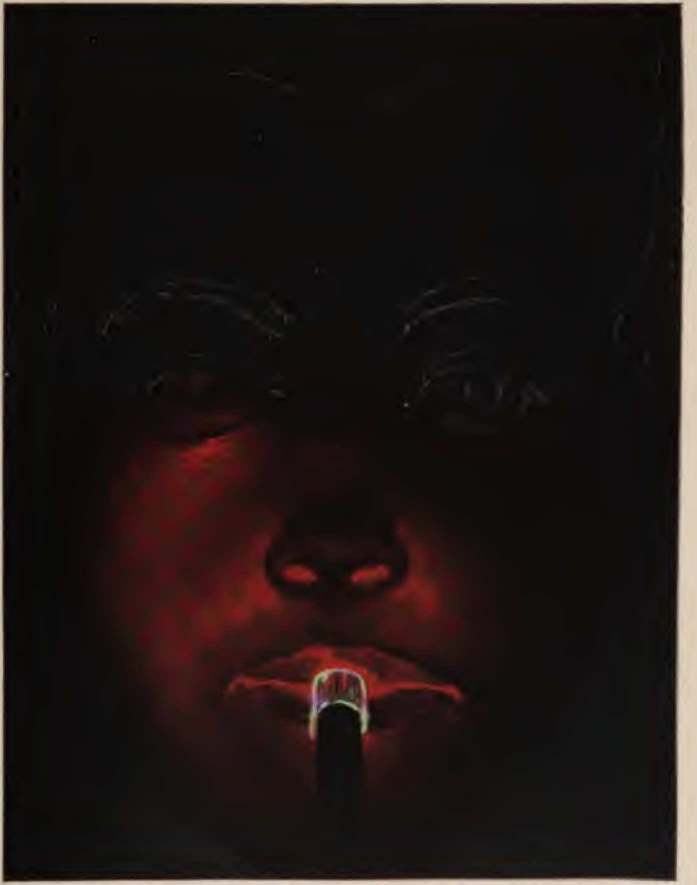
The test is carried out by placing a 10-volt electric lamp in the patient's mouth, darkening the room, and then comparing the degree of illumination of the two sides of the face, more particularly the regions immediately below the lower eyelids. When one antrum contains pus or a new growth, or has been the site of chronic inflammation, the normal crescent of light in the infraorbital region, and possibly in the cheek below it, will be absent, its place being taken by an opacity which is in marked contrast to the bright red, translucent appearance of the normal side of the face. The greatest opacity will be caused by a new growth filling the antral cavity; on the other hand, distension by a thin-walled cyst may cause abnormal translucency of the sinus.

The method is of little value in diseases of the frontal sinus, because it is impossible to prevent diffusion of light through the skin and tissues superficial to the sinus. It cannot be too strongly emphasised that while transillumination is a most valuable aid in the diagnosis of certain diseases of the nasal sinuses, the information derived from it is useful only in so far as it bears out suspicions which have been based upon other subjective and objective symptoms in the case. For fuller details as to transillumination, the reader is referred to the article on "Chronic Inflammation of the Antrum in Diseases of the Nasal Accessory Sinuses," p. 78, and to Plate I.

Skiagraphy.—Apart from their value in the detection of foreign bodies in the nose, the use of the *x*-rays is almost entirely confined to the investigation of chronic inflammatory lesions of the nasal accessory sinuses.

By the examination of a well-developed plate, the surgeon may be able to confirm suspicions, derived from other symptoms, that a frontal sinus or maxillary antrum contains pus, because its outline is hazy or ill-defined compared with that on the normal side. Furthermore, if external operation upon a frontal sinus be contemplated, the skiagram will give a very accurate idea of the size of the sinus, and therefore of the deformity which may result from operative interference. It may also demonstrate anatomical features which will have to be considered by the surgeon; for example, the presence of septa causing loculated spaces within the main sinus cavity; fronto-ethmoidal cells extending outwards below the floor of the sinus which will necessitate careful and very skilful treatment from the operator. Occasion may possibly arise when the surgeon cannot be sure whether his probe has entered the sinus he is desirous of examining; this can be ascertained without any doubt by

PLATE I



TRANSILLUMINATION OF THE ANTRUM.

The right antrum is normal and shews the well-defined infra-orbital crescent of light; the right pupil also exhibits the light-reflex. The left antrum contains pus, and the infra-orbital crescent of light is absent. (For this excellent figure I am indebted to my friend Dr. C. G. Coakley, of New York.)

means of skiagraphy. Finally, the presence of certain foreign bodies in the nasal cavities or the sinuses can be immediately detected by the examination of a well-developed plate. To obtain such a plate many details of technique must be observed; readers desirous of obtaining practical information on the subject should refer to the following two articles. HERBERT TILLEY.

BECK. "Radiography and Transillumination in Diagnosis of Sinus Disease," *Laryngoscope*, 1907, xvii. No. 11.—WASSERMANN. *Internat. Zentralbl. f. Ohrenh.*, 1907, v. No. 10.

H. T.

2. DISEASES OF THE NOSE PROPER

MALFORMATIONS OF THE NASAL FOSSAE

By GREVILLE MACDONALD, M.D.

Apart from the faulty development of the nasal fossae invariably associated, to greater or less extent, with the V-shaped superior maxilla, there is little to be said concerning malformations. In conjunction with narrow fossae there are almost invariably a high-arched palate, a short upper lip, with the septum variously deviated and thickened, and the vestibule often further narrowed by collapse of the alae. All the symptoms of nasal obstruction may appear as the consequence of such imperfect development, even without any of the forms of hypertrophy which so frequently aggravate the inconvenience of the narrow nose; though in the absence of chronic rhinitis in some form or of adenoids, the ears do not appear to suffer from a mere stenosis of the fossae. It is important to note that this stenosis, with the exceptions to be presently mentioned, implicates the anterior half only of the fossae, and that it is generally rectified by the correction of the septal malformations according to the methods described elsewhere (p. 47). Yet even when this is effected, the fossae not infrequently still remain too narrow to permit of easy breathing, and examination shews that, while the lower turbinals are perfectly normal and in no way hypertrophied, they are, nevertheless, relatively too large for the width of the fossae in which they lie. It is in such cases only that turbinectomy is ever justifiable; and even then it is seldom necessary to remove more than a portion of the anterior extremities. A more troublesome accompaniment of the narrow upper jaw and nasal fossae is found in a narrowing of the nostrils themselves, and sometimes in collapse of the alae. The former is congenital, and the latter results from chronic buccal respiration and disuse of the dilator muscles of the alae. Something can be done to correct the stenosis by patient stretching of the orifices with a finger; but the difficulty of the collapse can only be overcome by mechanical means for keeping open the nostrils. Of the various instruments devised for this purpose Dr. H. A. Francis's pattern is perhaps the best. But the patient is singularly intolerant of these during sleep, which is precisely when they are most needed; and a piece of rubber tubing, one-third of an inch long and of such calibre as will slightly distend the vestibule, is better borne.

The only condition of structural *stenosis implicating the posterior choanae*, apart from septal malformation, which is seldom or never congenital, is complete occlusion of one or both orifices by a membranous or osseous dissepiment. Such cases are fortunately extremely rare. They are always found in fossae faultily developed, though not, so far as I have observed them, in the V-shaped jaw, or necessarily in conjunction with the irregular septum. The latter, however, is abnormally wide posteriorly, encroaching upon and becoming adherent to the outer walls by membranous or osseous union. These cases are generally mistaken for adenoids, especially when the latter are associated with the occlusion. But the true nature of the case should be suspected when one or both fossae are found constantly filled with thin mucus which cannot be extruded, and when, moreover, it is found that no fluid can be driven by a syringe into the pharynx. When the occlusion is membranous, it is easily amended by incising the obstruction and removing with punch-forceps as much of it as possible. When osseous, the difficulty is greater, and may be insuperable; but here, small bone-forceps cutting at a right angle may effect much, and the ablation of the posterior extremity or the whole length of the inferior turbinal bone with the so-called spoke-shave may complete the cure. Of course such cases must be watched during cicatrisation so as to prevent readhesion. Occasionally in children, as well as in adults, we have to deal with cicatricial adhesions in the fossae. As they are sometimes impossible to explain as due to any definite cause, they may appear to be congenital. But they are probably always the result either of ulceration or of surgical interference. They may prove excessively troublesome; though free separation with the knife, and persistent watching and reseparation with a blunt instrument at the earliest appearance of readhesion, are very successful. Possibly in some of these cases fibrolysin might prove effectual.

GREVILLE MACDONALD.

RHINITIS

By GREVILLE MACDONALD, M.D.

ACUTE RHINITIS.—There are three varieties of acute rhinitis: (1) the catarrhal, (2) the dry, and (3) the croupous.

Etiology.—Among disposing causes must be mentioned chronic rhinitis, and stenosis of the nose, whatever its origin. Tuberculosis, syphilis, and hereditary tendency induce hypersensitiveness to sudden changes of temperature, exposure, and so forth. Anaemia is not of so much influence in this direction as might be supposed; but prolonged mental strain, muscular fatigue, and debility from sexual excesses do certainly induce susceptibility to catarrhs.

Besides cold-taking, whether infective or not, an acute rhinitis may arise from direct irritation. This is most frequently seen after exposure to the London fogs, or less often from simple exposure to a bleak wind after sitting over a warm fire; correspondingly it frequently occurs after the removal of some nasal obstruction such as polypus. Less often it is observed after the inhalation of irritating fumes, such as chlorine gas, the various acids and bichromate of potassium. In patients suffering from certain idiosyncrasies, ipecacuanha, iodine vapour, pollen-dust, and the like may induce aggravated symptoms. These idiosyncrasies are frequently associated with chronic inflammatory conditions. After exposure to some powerful local irritant the croupous form of inflammation is sometimes encountered.

(1) **Symptoms.**—In the *catarrhal* form the symptoms generally begin with soreness at the back of the nose. After a few hours there follows a watery discharge. At first the nose perhaps feels unusually free; but soon the sneezing and turgescence of the erectile tissue, which give to the attack its misery, assert themselves. The nose and lips become sore and swollen. As the swelling and obstruction increase, the sneezing diminishes and the discharge is more tenacious. Towards the termination of the attack it often becomes muco-purulent, and herpes often appears on the lips. The usual duration of an attack to which rational attention has been paid is three to seven days, though frequently, through neglect, the muco-purulent discharge persists for many weeks. Smell and taste are affected in severe cases, and may be absolutely abolished for some days.

If the frontal or other sinuses are implicated, there is fairly intense frontal headache or pain referred to the back of the eyes. When the patient complains of face-ache, the maxillary sinus is probably affected. Similarly there may be epiphora, or deafness and tinnitus from implication of the Eustachian tube.

In infants, in whom the nasal passages are peculiarly narrow, a very slight rhinitis will entail considerable respiratory disturbance; for it is only in older patients that mouth-breathing is discovered as a substitute to nose-breathing; and even when it appears that the mouth is open, the tongue may be closely approximated to the roof of the mouth. In such circumstances the child may be attacked with severe dyspnoea during sleep. Nor can he suck without distress. From the parents' accounts of these fits, as they call them, it would appear that laryngismus is sometimes induced.

Objectively, there is nothing to be seen beyond, in the early stages, a little dryness of the septum and inferior spongy body, and, in the later, intense swelling of the erectile tissue, with obliteration of the inferior meatus. The colour of the mucous membrane is intensely red and sometimes dusky, though seldom so dark as in chronic rhinitis.

(2) In those forms of acute rhinitis in which the *dryness* of the initial stage persists, the chief complaint is usually one of diffused headache. The sneezing is very slight, and there is little or no secretion from the nose. The senses of smell and taste are often quite abolished. Objectively, the most prominent feature is the swelling of the middle

turbinal, which appears dry and glazed. The inferior turbinal is not so conspicuously swollen as in the catarrhal form, while its surface also is dry, though not so distinctly as that of the middle.

The *treatment* is most successfully conducted on the general principles familiar to every household, though a full dose of quinine or salicin, and the inhalation of friar's balsam, may give great relief. Menthol as snuff or dissolved in paroleine, five grains to the ounce, relieves the sneezing and swelling. Cocaine and adrenalin give more striking relief, though their use is often followed by aggravation of the symptoms. A full dose of Dover's powder at bed-time, followed by small doses of quinine and opium in the day-time, often arrest an attack. But probably the most effectual of all remedies is the vapour-bath. (See also art. "Infectious Coryza," Vol. II. Part I. p. 570.)

With infants the difficulty of sucking when the nose is obstructed may necessitate taking the child from the breast and feeding it with a spoon; but a small rubber tube inserted along the inferior meatus will sometimes enable the child to suck easily.

(3) The **croupous** form of inflammation occasionally attacks the mucous membrane of the nose, and independently of diphtheritic infection. There is, of course, no reason why the Schneiderian should be exempt any more than the bronchial or intestinal or conjunctival mucous membranes. Here one cannot but recall the observations of Senator on the relation of croupous to diphtheritic membranes, the difference between them consisting merely in the different intensity of the inflammatory process. Had he ever witnessed the changes following the application of the actual cautery to the mucous membrane of the nose, the correctness of his comparison would have been even more convincing to him. In fact, the exudation produced after the application of the electric cautery and that formed in the course of an attack of croupous rhinitis are identical in all appearances, except so far as organisms are concerned. The Klebs-Loeffler bacillus is sometimes, though by no means always, present in croupous rhinitis; but its presence, or that of its innocuous mimicries, it must be remembered, may be found in the mucus of a perfectly healthy nose. There does not appear to be any special constitutional factor in the development of the affection. All that can be affirmed as to etiology is that one attack disposes to another (Potter). Probably climatic circumstances contribute to the etiological factors, since the disease appears to be more frequent in America than in Europe.

Clinically the *membrane* differs from the diphtheritic in that it is usually not adherent, and may be removed without producing any abrasion of the surface. Moreover, the mucous membrane may be, though greatly swollen, rather paler than in health; it may, in fact, be oedematous. But the character of the membrane is of not much value in diagnosis, the latter depending rather upon constitutional conditions. In the idiopathic croupous rhinitis the extent of the patch may vary from the size of a small pea to such a size that the whole of the nasal and naso-pharyngeal mucous membrane is covered with a thick, gelatinous but firm membrane, which may be withdrawn with forceps without any haemorrhage. On the posterior wall of the pharynx may be seen circular isolated patches lying on an unnaturally pale or inflamed base. These, though more adherent than in the nasal fossae, expose, when peeled off, a surface heightened in colour, and occasionally slightly haemorrhagic. Sometimes the affection is confined to the pharynx and naso-pharynx. Cocaine appears to have no anaesthetic effect, nor will adrenalin contract the swollen turbinated bodies in these cases. This anomaly I have often noticed in cases of sharp inflammation following the electric cautery or other operation; probably an inflamed surface has lost its power of absorption.

The *symptoms* are usually those of an ordinarily severe rhinitis. There is more or less occlusion of the nose with a profuse flow of watery mucus, pieces of membrane being occasionally extruded on blowing the nose. In some cases there is severe sneezing and less often pain. After the removal of any portion of the false membrane, there is a marked liability to its recurrence. In a patient under my care, who had had many attacks, there appeared at each attack a wide patch of erythema extending across the bridge of the nose. Schuler has noticed the association of a membranous rhinitis with facial erysipelas. The duration of the affection, as will be gathered from the preceding remarks, is very variable. Potter, who has recorded the largest number of cases, gives the average as three weeks. But occasionally cases run a chronic course.

The *diagnosis* from true diphtheria must depend upon bacteriological examination, cardiac and renal symptoms, and the sequels. Nasal diphtheria presents the gravest and unequivocal signs.

Treatment is of little help beyond the use of such remedies as are of service in ordinary acute rhinitis. Hot fomentations to the nose and eyes are of some use, while irrigations of weak solutions of borax have a soothing effect. The membranes, if extensive, should be removed as fast as they accumulate; and in cases in which they are so large that they cannot be extruded by blowing the nose, they should be removed by forceps. Potter found great relief from the introduction into the nasal fossae of dry tampons of cotton-wool. These, absorbing the excessive mucus swell and by their pressure on the erectile tissue, effect that reduction in size which cocaine is unable to accomplish.

The literature of the subject is not extensive, many of the cases recorded as pseudo-membranous rhinitis being obviously instances of diphtheria of the nose. Gluck has recorded some cases, Raulin has reported four cases of Moure, and Chapin has given us a good *résumé* of the subject. Lack (6) has written lucidly on the subject, and other writers have contributed to the literature of the affection (7) (*vide* also Vol. I. pp. 975, 998).

CHRONIC RHINITIS.—The subject may be arranged in the following tabular form:—

A. Catarrhal Rhinitis.—1. As affecting the erectile tissue:—(*a*)

Catarrhal rhinitis with vascular tumefaction of the erectile tissue, sometimes erroneously styled hypertrophic. (*b*) Catarrhal rhinitis with true hypertrophy and oedema of the erectile tissue.

2. As affecting the ethmoid bone:—(*a*) Hyperplasia of the ethmoidal muco-periosteum, developing into mucous polypus and cysts. (*b*) Caries and exfoliation of the bone, accompanied by suppuration, and generally involving one or more of the accessory cavities.

B. **Dry Rhinitis.**—(*a*) With simple mucous secretion. (*b*) With muco-purulent secretion, commonly called ozaena, or atrophic rhinitis.

Although this classification is based upon broad scientific data, there are often no hard-and-fast lines of distinction to be drawn. Thus, the middle and inferior turbinals are often affected together: the former may be hypertrophied, while the latter is collapsed. This condition again may be observed either in catarrhal or dry rhinitis, and so on. But if the main points in the classification are borne in mind, some common errors in diagnosis will be obviated.

The subjective symptoms are of small importance and no diagnostic value. Buccal respiration, snoring, inability to blow the nose, and the characteristic buccal speech are found in all forms of obstruction. The closed mouth and fetid breath; inspissated mucus hawked from the throat; the speech approximating that of the cleft-palate, are associated with abnormal patency of the fossae. The character of the discharge, apart from its degree of inspissation, whether mucous or muco-purulent, is of little value; but, when purulent or unilateral, it suggests suppuration of the accessory sinuses, necrosis, rhinolith, or a foreign body. The distinction between pus and mucopus presents no real difficulty; mucopus is always transparent, tenacious, and ropy; whereas pus, however thin, is opaque, and, however thick, is insusceptible of being drawn out into strings. The secretion, especially in children after some of the exanthemata, may be thin and acrid, when it may irritate the nostrils and upper lip. Pain is but seldom of importance, unless there is sinusitis, or muco-periostitis of the ethmoid. Cough is occasionally a prominent symptom, especially in children, and apart from any concomitant laryngitis. The nasal cough is dry and barking. Olfaction is of small value as a diagnostic, seeing that very insignificant causes may destroy it. Parosmia is common as a sequel to influenza.

A. **Catarrhal Rhinitis.**—1. As affecting the erectile tissue:—(*a*) *Vascular tumefaction* of the erectile tissue is the exaggeration of a physiological condition. It may become persistent and lead to serious inconvenience. One consequence is often overlooked—the imprisoning beneath the inferior turbinal of large masses of ropy mucus or mucopus, which the patient to his great distress cannot remove by blowing the nose, though relief is at once afforded by injecting any lotion beneath the anterior extremity of the lower spongy bone.

The etiology of this turgescence is not as simple as appears. We must suppose that diminished elasticity in the fibrous tissue around the venous sinuses prevents control of their distension by the arterioles.

Among the more important disposing causes must rank stenosis of the bony framework of the nasal fossae. In this state a very slight swelling of the erectile tissue will be sufficient to lower during inspiration the atmospheric pressure behind the point of stenosis to such a degree as will cause an additional overfilling of the venous sinuses. This tendency to simple chronic rhinitis with vascular obstruction is particularly common in individuals with large prominent noses, and in Jews; and in this type of nose there is considerable tendency to structural narrowness of the fossae. As exciting causes a moist atmosphere, sudden changes of temperature, or frequent acute attacks must be cited. Of local irritants the inhalation of fine dust appears to be often responsible for a chronic catarrh. Masons working in stone and marble are said to suffer but seldom; possibly the particles are too heavy to float in the atmosphere. On the contrary, the dust of alabaster is said by sculptors to be highly pernicious.

On examining the nose we find the field almost entirely filled with the swollen inferior turbinal, which, together with the like-conditioned floor and septum, completely obliterates the inferior meatus and encroaches upon the middle. By tilting the head well back we may generally obtain a view of the anterior enlargement of the middle spongy body. The points which distinguish this tumefaction of the inferior turbinal from other morbid conditions of obstruction are these: the prominent surface is smooth, uniform, globular; the colour is dark rose, sometimes purplish, though in anaemia, or when accompanied by oedema, as in those affected with paroxysmal sneezing, it may be grey or pinkish-grey. On examining it with a probe, we find that it pits very easily, the impression being immediately obliterated; it fluctuates as if, indeed, it were a sac but half-filled with its fluid contents. This impression on probing is quite pathognomonic. If further verification be required, a weak solution of cocaine, or highly diluted adrenalin, may be applied to the mucous membrane. These produce in a very few seconds a shrinking, and ultimately a complete collapse of the erectile tissue, so that the condition resembles that to be next described as the second variety of chronic catarrhal rhinitis. In determining exactly whether or no the inferior turbinal is too much engorged, it must be remembered that frequently in cases of deflected septum the erectile tissue is unusually large on the side of the concavity. In such cases it must not be considered as necessarily pathological, except in so far as it may be compensatory to a pathological condition.

As coexisting conditions there is often a catarrhal, sometimes hypertrophic, inflammation of the naso-pharynx, the pharynx, and larynx. The tonsils in children are sometimes enlarged, and post-nasal adenoids are common. Within the nose itself there may be every condition and stage of chronic inflammation of the various structures, especially enlargement of the anterior extremity of the middle turbinal, and hypertrophies of the septum, cartilaginous and osseous.

There is apparently little tendency to spontaneous cure; on the contrary, there is sufficient clinical evidence to shew that the disease advances towards true hypertrophy. The prognosis is more favourable in those cases in which there is a narrowing of the spaces from hypertrophy of the septum, provided this can be remedied.

Treatment.—This is simple enough. In recent cases, especially those associated with a naso-pharyngeal catarrh, it may be sufficient to prescribe a simple detergent wash for the nose, such as ordinary Dobell's solution. Cocaine and adrenalin should never be used as remedies. In spite of their temporary relief they both aggravate the disease. If the patient be unable to sniff up the lotion from the hand, it may be run into the nose by the help of a syringe or glass tube; or it may be sprayed into the nose, though this is hardly as effectual. Some prefer the syringe, in which case it should be directed along the inferior meatus, with the nozzle of the instrument pointing obliquely downwards. In any case it is essential that the fluid be drawn into the throat. The nasal douche is objectionable, because the high pressure and the bulk of fluid engender some risk of the fluid entering the Eustachian tubes. In cases in which ropy mucus persistently lies beneath the lower turbinal, it is only a skilled hand that can effect thorough washing. In these cases a fine curved nozzle must be introduced directly beneath the anterior extremity of the body and the retained mucus washed vigorously away. What the dentists call a chip-syringe is admirable for this purpose. As curative agents, drugs appear to be of little or no use, so intolerant is the Schneiderian membrane of even the weakest astringents. Menthol is harmless, though its benefit is but temporary in relieving turgescence.

Finally, in the way of radical cure, we may have recourse to linear cauterisation of the swollen tissues with the galvano-cautery, or to a limited use of chemical caustics. In order to produce contraction of the tissue filling the concavity of the inferior turbinated bone, a probe, upon which is fused a small crystal of chromic acid, may be passed over the swollen surface. This is more easily limited in its application than the monochloracetic or nitric acids. It also causes the least pain, though the monochloracetic acid has the advantage of forming a slough which is retained until cicatrisation is completed beneath it, thus obviating the danger of adhesion should opposite sides be inadvertently touched with the reagent. Superfluous acid should be immediately removed by a piece of wool wrapped round a second probe, or should be washed away with an alkaline solution. In employing the electric cautery a simple burner is preferable to any guarded instrument, as it allows more perfect vision. It should be heated to a cherry-red. If too hot, it is apt to produce haemorrhage, and if of only a black heat it may be decidedly painful, even when the surface is cocainised. Before employing any cautery measures, a 5 or 10 per cent solution of cocaine should be carefully rubbed into the affected area with a pledget of cotton-wool. Specialists have long discarded the spray, and eucaine or alypin are often preferred. The latter is perhaps the better, and may be used in a 20 per cent solution. It appears to be quite harmless, and, combined with a little adrenalin, the hyperaemia its application induces is avoided.

So satisfactory are these lines of treatment in most cases that, where they do not succeed, the failure is probably due to some hypertrophy or chronic oedema. But of course the general health must always be attended to. Where there is much dryness, a stimulating wash, such as three grains of ammonium chloride to the ounce, gives considerable relief.

(*b*) *Catarrhal Rhinitis with true Hypertrophy of the Erectile Tissue.*—This condition implicates chiefly the inferior turbinated body, though in rare instances it affects the floor and septum to such a degree that the hypertrophies have been actually described as polypus and papilloma—mistakes not inexcusable, considering the occasionally close resemblance.

Etiology.—The condition is probably due to a persistence of engorgement, as in the less serious cases engorgement is always associated with hypertrophy. It is most frequent in patients whose noses have narrow fossae. Like polypus, the affection is sometimes distinctly hereditary; the only history obtainable is of a succession of colds in the head, the affection apparently only reaching the stage of hypertrophy after the lapse of many years. It is found chiefly in males of all ages, though the older they are, the more severe, as a rule, is the hypertrophy and its symptoms; some very severe cases, however, are occasionally met with in young adults of both sexes, and then nearly always associated with post-nasal growths. Persistent obstruction in a child after adenoids have been successfully removed, is generally due to hypertrophy of the turbinated bodies or to malformation of the septum.

The pathology of this hypertrophy is simple enough if it be considered as the result of simple hypernutrition of pre-existing elements combined with inflammatory action. For there is marked overgrowth of the venous sinuses as well as of all the connective-tissue elements and the lymphoid tissue. The only point in which these differ from the normal is the separation of the elements by an oedematous exudation. The cauliflower form is conspicuous for the large quantity of small-celled growth embedded in a fine reticulum of connective tissue; whereas, in the smoother, darker, and less lobulated forms, particularly those which project into the naso-pharynx, there is a general hypertrophy of all the elements—erectile, connective, and adenoid tissues, and even of the mucous glands. Why the hypertrophied mass should assume a dendritic arrangement is not so easy to determine. But it is interesting to remember that this is one of the forms adopted by nature for increasing the area of a secreting surface, namely, by a process of protrusion instead of the more usual inversion.

Symptoms.—A well-marked case presents all the symptoms of chronic rhinitis in a greatly aggravated form. The discharge is generally profuse, sometimes very tenacious and slightly purulent, and there may be all the usual symptoms of pharyngeal and laryngeal catarrh. Cough is occasionally troublesome, not only from the accompanying laryngotracheitis, but as the result of reflex irritation in the nose. Though the palate is often practically paretic, as judged by its pendulous attitude and irresponsiveness to tactile stimulation, the voice retains the characteristic quality of nasal obstruction.

Examination of the anterior nares shews first a quantity of ropy mucus hiding the structures, so that if the patient be unable to blow through his nose, it may be necessary to clear it with a syringe; in the worst cases this reveals a pale, oedematous mass, obscuring the normal structures, and, at first sight, closely resembling an ordinary mucous polypus. On closer investigation, however, it will be seen that the mass is distinctly, sometimes finely and deeply, lobulated, with perhaps a cauliflower appearance. Examination with a probe brings out this arrangement more satisfactorily, and, moreover, shews that it is in all probability attached to the inferior turbinal, or if not, as rarely happens, to the lower portion of the septum, or to the floor of the nose. In cases of exceptional duration, such masses may spring from quite high up on the septum, and are then generally associated with real polypus. Wherever situated, the mass may be freely movable upon its attachment, and it may be fixed by a wide base. It is never connected with the middle spongy body, and never descends from the superior meatus. When cauliflower-like, it often involves the whole length of the inferior turbinal, filling the inferior meatus, and closely packing the concavity of the bone; so that after the passage has apparently been cleared by operative measures, it may still be possible to turn out of this recess large quantities of the growth. More rarely these growths are rounded and solitary, assuming almost a pedunculated aspect; or there is but a finely lobulated fringe attached to the lower margin of the inferior turbinal. But the anterior portion of the hypertrophy does not always assume the pale cauliflower appearance; it may be pinkish or rose coloured, and very coarsely lobulated. In such cases there is some engorgement of the venous sinuses in addition to the genuine hypertrophy. In this case the probe reveals a marked tendency to pitting, whilst cocaine will induce a partial shrinking. By this means the relative amount of hypertrophy and turgescence will be precisely indicated. Indeed, without cocaine or adrenalin, it may be difficult to determine the right methods to be pursued for treatment. The transition between the two states is sometimes ill-defined; but whenever there is the slightest appearance of lobulation, the condition must be considered as hypertrophic and treated accordingly.

On examining the posterior nares, the same sort of growth may be seen covering the inferior turbinal, projecting into the post-nasal space, and even weighing heavily on the soft palate, causing it to assume, more conspicuously than in any other affection, the perpendicular position. Nevertheless, there is this difference in the appearance as seen from before and behind: posteriorly the hypertrophy, having more room for increase in size, has far less tendency to minute lobulation, and may even, though rarely, appear perfectly smooth. In this case, if oedematous, it may be indistinguishable in appearance from polypus; while, when pink or pale rose colour, it is sometimes mistaken for post-nasal adenoid growths. Perhaps it is upon the evidence of such cases, and when the turbinal hypertrophy is associated with adenoids, that some authorities describe the latter as growing from the septum or other parts. As a further variation, the projecting portion may be smooth circumferentially, but finely lobulated in its central, most prominent region. Occasionally the growth in a backward direction is so enormous that it completely fills the post-nasal space. In this contingency it is only by digital examination that the point of origin of the growth or growths can be determined, and the diagnosis from adenoids or polypus be established. Sometimes, when the posterior enlargement is chiefly due to oedema, and is yet sufficiently pronounced to be seen in the post-nasal mirror, its structure is so soft and yielding that it is almost impossible to detect it with the finger. This is a point to be borne in mind when attempting to remove it with the snare. In rare cases the hypertrophied posterior ends are of a dusky rose colour, almost purple, distinctly granular or evenly lobulated, and may be as large as a small walnut. The appearance has been aptly likened to that of a ripe mulberry. They are usually symmetrical, and to the finger give an impression of considerable substance.

The most frequent associated affections, besides those so often attendant upon all forms of nasal obstruction, are hypertrophies and deflections of the septum, enlargement of the middle turbinal, and hypertrophy of the post-nasal adenoid tissue.

The diagnosis of the affection presents no difficulty in the light of the preceding description. From polypus it is distinguished by lobulation and firmer attachment, as well as by the region from which it springs. The usual smoothness of a polypus is modified only when its surface becomes papillomatous, a very rare change. In tuberculous disease of the nose the proliferating granulations might possibly be mistaken for oedematous hypertrophy of the erectile tissue; but the purulent secretion in the former, with the friable and haemorrhagic nature of the obstructing growth, should leave no room for doubt.

The prognosis is decidedly good if the patient is willing to submit to surgical treatment; but there is no tendency towards spontaneous cure. I have seen the nose tightly packed with this lobulated growth in a Jew of seventy-five, who said his nose had been obstructed all his life; yet there was no difficulty in effecting a cure, and relieving the reflex cough for which alone he had sought relief.

Treatment.—The only methods of treating this hypertrophy are surgical, although temporary relief is given by syringing. The means almost universally adopted is the snare. Of the various instruments used for this purpose, that invented by Jarvis, who initiated this method of operating upon the nose, is probably the safest. The special point of superiority in his instrument for these growths is the slowness with which the loop can be tightened, and the slighter risk of bleeding, which may otherwise be very profuse. I have devised a modification of it, the additional merits being a handle by which it can be firmly held without obstructing the field of vision, and a collar with four radiating spokes, which, one by one, can be turned with the thumb of the same hand, and the screw thus slowly withdrawn with its attached wire. Thus the left hand is free for holding the speculum or inserting the forefinger into the naso-pharynx. Most operators prefer to remove the posterior hypertrophies, as well as the anterior, with a local rather than a general anaesthetic. When a local anaesthetic is used, it is often possible, from the paretic condition of the soft palate in this affection, with the assistance of the post-rhinoscopic mirror to hitch the snare, passed through the anterior nares, over the neoplasm—a proceeding in every respect pleasanter to the patient than fixing the noose with the forefinger in the naso-pharynx. A general anaesthetic, however, is often needed.

B. **Dry Rhinitis.**—(*a*) *Rhinitis sicca*, in which the secretion consists almost entirely of mucus, appears to be entirely distinct from the forms of chronic rhinitis already described. It is found for the most part in overfed persons of middle life, whom there is often reason to suspect of alcoholism. The patient complains of a dry, cobweb-like feeling in the throat, of huskiness or actual aphonia, or of deafness and fulness in the ears; he seldom needs a pocket handkerchief, but hawks thick phlegm from the throat. Sometimes he produces hard crusts, which, he says, he has coughed up, but, from their dome-like configuration, have evidently been moulded in the vault of the naso-pharynx. The process of inspissation may extend not only into the pharynx but even into the larynx and windpipe, and the patient may actually expectorate greenish-black crusts which have been accumulating there for several days and sometimes impeding respiration. A patient in this plight may be excusably supposed to be asthmatic; and, before the extrusion of the offending mass, there may be some real anxiety from dyspnoea. Crusts collect in the anterior nares and especially on the septum; these the patient is apt to remove with the finger, thus producing excoriation and epistaxis. As a further consequence there may be ulceration, sloughing of a larger or smaller portion of the triangular cartilage, and perforation. When such a mishap results from simple inflammatory conditions, the destruction never involves the osseous septum. There is no special fetor in the patient's breath; this distinguishes these cases from ozaena, syphilitic necrosis, and other conditions.

The rhinoscope may shew some increase in the lymphoid tissue on the posterior wall, sometimes in the form of two folds running vertically on each side of the median line, growing more prominent as they ascend and merge into Luschka's tonsil. In the central furrow thus formed the mucus may cling very tenaciously. This condition is one of those described by Tornwaldt of Dantzig in 1885 as a peculiar disease. But there is no clinical warrant for admitting "Tornwaldt's disease" into our nomenclature.

Diagnosis.—It must be remembered that general anaemia is often accompanied by collapse of the erectile tissue and diminished secretion of mucus, and that without any chronic rhinitis there may be a general dryness of the pharynx as well as of the anterior nasal region.

In the treatment of simple dry rhinitis, a course of Carlsbad water will do more than local applications. The best lotion for cleansing is a non-stimulating solution of borax and bicarbonate of sodium, or the ammonium chloride lotion. When large crusts form in the naso-pharynx it may be necessary to wash them away with a post-nasal syringe. For the dryness in the throat a carbolic acid lozenge, or a borax pastille, may give great relief. For the laryngeal and tracheal dryness an inhalation of creasote may prove of advantage; but if there be actual crusts in these regions, an alkaline or sodium chloride spray, inhaled for five or ten minutes as often as necessary, may be required to detach them.

(*b*) *Atrophic Rhinitis, or Ozaena.*—The large majority of patients suffering from this disease are children and young adults. The affection usually dates from an attack of measles, scarlet fever, or other exanthem responsible for a chronic muco-purulent rhinitis; from one or from a series of bad colds in the head; from a severe blow on the nose, followed by epistaxis; or, lastly, from unassigned causes, such as inherited or tertiary syphilis. It occurs, for the most part, among the poor, though it is by no means uncommon among the prosperous. In almost all cases of long duration there is considerable anaemia; the local disease being apparently responsible for the constitutional condition. Young domestic servants appear to be peculiarly liable to the affection. A certain proportion of the patients present the characteristic so-called "strumous" physiognomy: the nose is small, the bridge and alae wide, the point of articulation with the frontal bone much depressed, and the nostrils look forwards and downwards. The nose sometimes appears to be sunk into the face, as it were, so that the cheeks and upper lip rise up from the attached margins of the nose. Even when the patient does not present this characteristic appearance there is generally an unusual width of the nose.

Pathology.—No new information has been offered since the first edition of this work. All agree that the place of the ciliated epithelium is taken by squamous; according to Krause: "The most remarkable thing is the presence of an enormous infiltration of round and spindle cells, which do not shew anywhere a distinct contour, but have disintegrated into a fatty detritus, which is often still arranged in the original cell form; and besides this infiltration, the presence in the mucous membrane of numerous large and small fat globules. The enormous quantity of fat secreted into and beneath the epithelium will quickly undergo disintegration, and change into fatty acids in the crusts, and will thus produce the sickening and rancid smell." Hajek, who made a bacteriological investigation of the disease, considers the large numbers of bacteria found in the secretions to be responsible for the fetor, though their absence from the mucous membrane proves that they have no etiological connexion with the disease. He found a constant and specific organism, the *Bacillus fetidus*, the culture of which yields the characteristic odour of ozaena. Löwenberg has described an organism like the pneumococcus, which he considers as specific. It is supposed, especially by the German school, that the atrophy is a further stage of a condition of hypertrophy, though on what grounds it is difficult to surmise (MacDonald). An ordinary catarrhal hypertrophic rhinitis never leads to an atrophic, except perhaps in the presence of caries; while the simple dry rhinitis, with engorgement of the venous sinuses, can never lead to ozaena, as may be inferred from the essential points of distinction between the two diseases. That in atrophic rhinitis there is no ulceration is admitted by all who have made autopsies in these cases. The only possibility of ulceration is the accidental one resulting in excoriation of the septum from picking the nose. There is very general consent as to the fibrotic condition of the mucous membrane, the attenuation of the bones, and the diminution of the blood-vessels; while the glands, both those of Bowman and the racemose, are diminished in number, and present distinct evidence of fatty and granular degeneration. The complete absorption of bone and mucous membrane is of great pathological interest. Possibly the conversion of protoplasm into fat, as found in Krause's and Habermann's autopsies, is the result of anaemia, and the initial step in the absorptive process. Further, we may remember that fatty degeneration and absorption of bone coexist in the disease known as osteoporosis, and that complete absorption of bone is known to occur without any apparent inflammatory or degenerative changes.

Symptoms.—Advice is usually sought for the foul breath which may make the patient's presence, even when at a considerable distance, almost unbearable. Hence the patient soon learns to shun society. There is generally complete anosmia. At varying periods large crusts are expelled from the nose, and great relief follows, while the odour is for a time less intolerable. The fetor is so peculiar that it needs no description; the only smell approximating it in intensity is that accompanying syphilitic necrosis in the nose; but though this is equally offensive, it is quite different.

On examining the interior of the nose the first noticeable point is the absence of vibrissae. Insertion of the speculum at once shews the essential feature in the disease, the abnormal patency in the nasal fossae; so widely are the turbinals separated that it is always possible, after cleansing the nose of obstructing crusts, to obtain a view of the posterior naso-pharyngeal wall, and frequently of the Eustachian tubes. There is a wide cleft between the middle turbinals and the septum, unless the latter presents an obstructing ridge, or the middle turbinals are enlarged from hyperplasia. Through the olfactory fissure the superior turbinal may be seen posteriorly. The inferior turbinals always appear small from collapse of the erectile tissue; not necessarily from atrophy, as the results of treatment occasionally prove. Cases of long duration shew either true atrophy of both inferior and middle spongy bones, or atrophy of the former associated with hyperplasia of the latter. In advanced cases both middle and inferior turbinals may have disappeared entirely. In such truly atrophic cases the opening into the naso-pharynx appears like a comparatively small orifice, above which may be seen the anterior surface of the sphenoidal sinus, looking downwards and forwards. In some cases it would appear that the walls of the ethmoidal cells must have atrophied and thus contribute to the large cavern into which we look. Examination of the pharynx and post-nasal space reveals the same general conditions of dryness of the mucous membrane, especially over the posterior wall, which is coated by a film, varying in depth and consistence, of inspissated mucopus. In the more severe cases there appears to be shrinking even of the muscular structures; while the soft palate may be so attenuated that the speech resembles that characteristic of the cleft palate. But there is hardly ever such complete atrophy of the turbinals behind as is seen from the front.

In an early stage of the disease there is no atrophy, although the fossae may be abnormally wide. Consequently, seeing that this roominess is invariably present, even when there is no atrophy, the question arises whether it may be responsible for the subsequent train of symptoms. The only possible influence that this roominess could have upon the functions of the nose is that of facilitating the passage of air at the expense of the velocity of the current. As a result, blowing the nose is less efficient in clearing it, and putrefactive changes and fetor will of necessity follow. Moreover, putrefaction means irritation, and the discharge becomes increasingly purulent. This irritation may induce an inflammatory hypertrophy of the middle turbinal, involving both bone and mucosa; and, by an infective process, it may lead to suppuration of the ethmoidal cells or of the maxillary sinus, and perhaps even of the sphenoidal sinus. Indeed, the association of atrophic rhinitis and suppuration of the accessory sinuses is so common that atrophic rhinitis appears to be an initial factor. Be this as it may, the inspissated secretion contracts much like a film of collodion, and causes anaemia of the subjacent mucous membrane. The immediate result of this is the collapse of the erectile tissue, and the opening up of the inferior meatus. This production of anaemia by surface-compression may also contribute to the ultimate atrophy of the structures, and possibly to the absorption of the osseous tissue. Moreover, a similar collapse of the erectile tissue is sometimes observed in other conditions. After removal of nasal polypi, for instance, in cases in which the inferior turbinals had previously presented a normal appearance, there is sometimes induced a muco-purulent discharge, which, partly from its tenacity, and partly on account of the unnaturally widened fossae, the patient is unable to extrude. It putrefies and produces an ozaenic smell; it dries and contracts the venous sinuses of the erectile tissue. A vicious circle is instituted, which, if neglected, might presumably result in true atrophy. Indeed, the removal of polypi has been quoted as a cause of ozaenic rhinitis (Schech), and I have observed the symptoms of ozaena with collapse of the inferior turbinal supervene after the same cause. Routier has recorded a case in which ozaena followed the eradication of a nasal sarcoma. The hypothesis of abnormal patency may therefore be regarded as correct in so far as it induces a tendency to stagnation and putrefaction of the mucopus. In certain cases in which the atrophy is unilateral, we almost invariably find an osseous spur blocking the other nasal fossa. Nevertheless, in the face of the frequency with which true ozaena is found without atrophy, it is impossible to endorse the hypothesis that the primary factor is deficient development of the turbinals (Gottstein, Zaufal, A. Hartmann), or to share Zuckerkandl's view that the initial trouble is turbinal atrophy.

Diagnosis.—Beyond the distinction between the two forms of dry rhinitis there are certain other points in diagnosis to which attention must be drawn. From tuberculosis there may occasionally be some difficulty in diagnosis. The crusts in lupus vulgaris may resemble those of ozaena, but occur on the septum in preference to other regions. They adhere very closely, and can scarcely be removed without haemorrhage; after their removal the presence of ulceration or of the characteristic tubercles immediately makes the diagnosis certain. In the more acute forms of tuberculosis the large masses of pale granulations coexisting with superficial ulceration will prevent any error. There may possibly be some confusion between the stench of syphilitic necrosis and that of ozaena, but in every syphilitic case a probe will reveal the presence of a sequestrum. Yet it must be remembered that conditions exactly resembling simple ozaena, as regards crusts, atrophy, and smell, may occur in the course of hereditary or tertiary syphilis, or may apparently be induced after the eradication of the constitutional ailment.

In suppuration of one or other of the accessory cavities it will be sufficient to remember that the discharge is usually unilateral, has little tendency to incrustation, and makes its exit spontaneously; that the fetor when present is more conspicuous to the patient than to the bystanders, and that sense of smell is not materially affected. With rhinoliths also the discharge is unilateral and fluid, though sometimes very offensive. Examination with a probe will reveal their presence.

The prognosis of ozaena is satisfactory, notwithstanding the assertion of most authorities that the disease is incurable. Of course, regeneration of atrophied structures cannot be expected, although the refilling of collapsed erectile tissue, if not in a state of advanced atrophy, is often observed. In early stages a real cure may be effected, while in the majority of even bad cases a symptomatic cure may be expected. Since it is rare for patients to seek relief after the age of thirty-five, it appears that the condition tends to improve spontaneously, if not to become cured, possibly because of the opening up and freer drainage of the sinuses. The function of smell, however, is rarely restored.

Treatment.—In spite of the innumerable remedies advocated, the treatment is simple, its success depending not upon the remedy employed, but upon the manner in which it is used. The only object is to secure cleanliness. For this purpose the nose must be thoroughly and repeatedly washed, and that by the practitioner himself; for herein lies the whole secret of success. In the initial stages of treatment it is almost impossible for the patient or her friends to effect any amelioration. The nose must be washed at least every day, and in such a manner as to remove every particle of putrefying pus, so far as the crusts come within the field of vision. This is followed by the utmost relief to the patient and her friends. Nothing acts more satisfactorily than the normal saline solution, though, for our own comfort, some hydrogen peroxide or sanitas may be added. It is remarkable how ineffectual is haphazard syringing; and it is only by the help of the speculum and infinite patience that success will be won. Occasionally a cotton-wool mop will help in removing the semi-detached crusts. Beyond the cleansing of the nose, we must seek to prevent further drying of the secretion; this is best effected by the method devised and advocated by Gottstein. This consists in the insertion into the nasal fossae of a tampon of cotton-wool, so as to fill completely the widened inferior meatus. The wool should be non-absorbent, so as not to abstract the mucus when it begins to flow. There is no question as to the great benefit resulting from this mode of treatment. Practically it is unnecessary to insert the wool for any distance, it being quite sufficient to plug the nostrils. This, moreover, is of advantage in that the patient can herself change the tampon as often as may be necessary. Some practitioners think it advisable to employ wool variously medicated, as, for instance, with iodoform, boric acid, ammonio-chloride of mercury. These may possibly be of some use, although it is hardly appreciable when associated with the immense benefit accruing from thorough cleansing and prevention of inspissation. It has recently become an accepted line of treatment to narrow the inferior meatus by submucous injections of paraffin (Lack). Though this can bring no restoration of function to the atrophied bodies, it permanently reduces the abnormal patency of the fossae, and this greatly facilitates the cleansing of the nose.

Beyond the use of such simple stimulants as the chloride of ammonium wash, or the medicated wools, remedies are worthless. But glycerin, with which a tampon should be saturated, is probably an unobjectionable stimulant, and acts very satisfactorily. Boroglyceride also, used in a similar manner, may prove very helpful.

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G. M.

EPISTAXIS

By Greville MacDonald, M.D.

Epistaxis is due to local or remote causes. The latter are such as induce, for one or another reason, alterations in the blood-pressure, namely, valvular disease of the heart, kidney disease, and arteriosclerosis. In emphysema also and in whooping-cough there is a liability to nose-bleeding; while nasal obstruction, especially that of adenoids, is often responsible because of the alteration in the blood-pressure induced by deficient oxygenation. Changes in the composition of the blood, such as are due to alcoholism, hepatic cirrhosis, haemophilia, anaemia of all kinds, scurvy and purpura, may induce epistaxis; and it is not infrequent as a premonitory symptom in the exanthemata, or in the course of enteric or diphtheria. It is occasionally vicarious for menstruation, and rarely alternates with haemoptysis in the early stages of pulmonary tuberculosis. High altitudes and tropical heat frequently induce it; while it has been noted in poisoning by phosphorus and some other drugs.

Morbid Appearances.—Of local conditions, that most commonly seen is a curious condition of the mucous membrane covering the cartilaginous septum, together with the adjoining floor and opposite area of the inferior turbinal. In this state the epithelial surface becomes attenuated and friable, the subjacent perichondrium infiltrated with small round cells, and the blood-vessels, capillaries, venous radicles and more rarely arterioles, from changes in their walls rupture upon the slightest provocation. This condition is accompanied by dryness on the surface, from atrophy of the mucous glands, and inspissation of mucus, which collects in crusts that necessitate removal with the finger. .Then follows a superficial excoriation and epistaxis, succeeded by ulceration which may lead to necrosis of the cartilage and perforation of the septum. Indeed it seems highly probable that many cases of epistaxis really originate in the juvenile habit of nose-picking. On examination of the nasal fossae, the glazed condition of the anterior part of the mucous membrane covering the triangular cartilage is very apparent. The line of demarcation between skin and mucous membrane becomes wider and less defined, and in old-standing cases a white cicatricial epithelial covering may extend upwards and backwards for perhaps an inch or more. In this case the tendency to haemorrhage has become spontaneously arrested. In others the mucous membrane becomes redder, thicker, softer, so that the patient can hardly so much as wipe the nose without disaster. Occasionally, even after complete obliteration of bleeding spots on the septum, epistaxis is not arrested, and we find that a larger vessel, artery or vein, has ruptured on the floor of the fossa. Such a point is occasionally hidden in the angle between the free border of the lower spongy body and the septum. Or again, the bleeding-point may be quite out of reach on the posterior aspect of a vertical spur, and the bleeding cannot be effectually stopped until the obstruction is removed. A case has been recorded in which the haemorrhage was traced to the naso-palatine artery (4). The loss of blood may be trifling or very profuse, so that in a few hours the patient becomes blanched. It must be remembered that this local cause for the haemorrhage does not preclude a constitutional trouble from being primarily responsible; and I have seen the same condition of the septum in old people suffering from arteriosclerosis, in chronic alcoholism, and in enlargement of the liver.

Other local conditions besides this degenerate condition of the mucous membrane of the septum, which may be met with in patients of almost any age, temperament, and habit, are found in certain specific forms of ulceration, such as tuberculosis and syphilis. Rarely an angio-granuloma, springing from a healthy mucous membrane and generally situated high up on the septum, will bleed very profusely, and only a skilled examination will detect this often minute neoplasm; it is never as large as a split pea, is sometimes pendulous, and does not recur after thorough removal with curette or cautery [cf. p. 53]. An extremely interesting group of cases in which epistaxis may be a prominent symptom is that of multiple hereditary telangiectases; Professor Osler has collected notes of eight families with this condition. Repeated epistaxis occurs from angiomas on the nasal mucosa, especially of the septum.

Sometimes, it must not be forgotten, epistaxis is due to traumatic causes of trifling or grave moment. A haemorrhage may result in pugilists from a mere sudden bending of the triangular cartilage or from a fracture of the base of the skull. So that every case of bleeding following a blow on the nose should be immediately and carefully examined, if only for the sake of rectifying a fractured septum. Epistaxis too is often the first symptom to draw attention to the presence of malignant growths in the nose or naso-pharynx. When accompanied by much sleepiness and the usual signs of nasal obstruction, its occurrence is highly symptomatic of those slowly growing sarcomas in young adolescents which were first described by Nélaton as fibrous tumours of the naso-pharynx. Rarely only is nose-bleeding found in polypus, though, of course, it is common in all forms of malignant disease. I found that an epistaxis of many years' duration arose in the sphenoidal sinus, being probably due to a carious leaking of the cavernous sinus: an opinion that was subsequently substantiated by the production of an orbital aneurysm. Epistaxis in slighter degree may occur wherever there is granulation tissue.

Epistaxis is, less often than is generally supposed, accompanied or preceded by general symptoms. Sometimes in the full-blooded, or those who suffer from "determination of blood to the head," nose-bleeding is ushered in by headache and is welcomed as a certain relief to the symptoms. But quite as often, more particularly after a severe attack, headache follows rather than precedes the attack. Sometimes tinnitus is relieved by a nasal haemorrhage, and sometimes it is symptomatic of the subsequent anaemia. Indeed the "fulness of the head and singing of the ears" in women at the menopause is sometimes, in my experience, relieved wonderfully by an artificial letting of blood from the inferior turbinals.

Treatment.—In most of such cases the haemorrhage will cease spontaneously or respond to the domestic cold key down the spine, raising the hands above the head, putting the feet in hot water, and so forth. But not infrequently the occurrence of the attack at short intervals or its severity demands the best skill and patience of the rhinologist. In almost every case one or more bleeding points can be found and their vessels obliterated with the electric cautery. Yet only too often the bleeding breaks out afresh on the slightest touch in the application of cocaine or adrenalin; and it may be so profuse as to allow no time for absorption of the solutions. But with patience and adrenalin there should be no difficulty in anaesthetising the surface and applying the cautery point; the latter should be heated only to a dull red and buried deeply into the tissues. Every angle and corner must be religiously searched for bleeding vessels. Sometimes the bleeding surface is so extensive and friable that the haemorrhage is rather like a general and rapid oozing than a flow from definite points; and more than once I have been compelled to curette the septum over an extensive area and down to the cartilage, so as to substitute a firm cicatrix for a spongy surface. And whenever this is necessary it is, if severe, absolutely satisfactory. After cauterising it is advisable to keep the affected area moist with a semi-fluid ointment, so as to prevent inspissation of mucus over the eschars and the possibility of the latter being removed prematurely. Indeed it should be remembered that generally ten days are occupied in cicatrisation after such use of the electric cautery. A useful prescription is the following: acid. boric. ʒss., menthol gr. v., adipis lanae ʒss., paraff. fluid. ad ℥i. The older styptics are now entirely supplanted by adrenalin and hemisine. Formerly I was in the habit of recommending the use of finely-ground matico leaves as preferable to any others. Ice on the nose and brow, or the injection of ice-water into the fossae, will generally stop the bleeding, at any rate temporarily. Or the injection of water at 110° F. is sometimes efficacious. Perchloride of iron should never be used; but a saturated solution of calcium chloride is very effective. The danger to the middle ear of posterior plugging is fully realised by all aurists and rhinologists (4). Out of many hundreds of cases of nose-bleeding that I have been called to treat, I have never once needed to plug the posterior nares, seeing that the bleeding almost invariably occurs, unless due to injury or malignant disease, from the anterior regions of the nares. Consequently tight plugging—and generally the firm grasping of the nostrils in the fingers—will almost always temporarily arrest even alarming bleeding. The many mechanical devices for plugging, such as rubber air-bags, water-bags, and ice-bags, are entirely superfluous. The position of the body is of some importance as affecting the flow of blood from a ruptured vessel. The veins of the nose, including those of the erectile tissues, pass for the most part into the pharyngeal plexus. Consequently the position of the head generally adopted, namely, that of stooping over a basin, is the most likely to favour the bleeding. On the contrary, the patient should be made to lie on the back with the head and shoulders partly raised, so that the blood accumulating in the pharynx can be easily spat out by turning the head on one side.

The treatment is somewhat different when the haemorrhage is due to causes affecting the blood-pressure or the coagulability of the blood. These must be treated on general principles. Saline aperients are probably helpful in all forms of epistaxis, and the lime salts are of signal use even when there is no history of haemophilia. When alcoholism or enlarged liver is the primary causes, the bleeding may be alarmingly profuse and plugging may be difficult. In such cases it sometimes appears that the ruptured vessels are higher in the ethmoidal region than can be discovered or reached with the cautery point; and we have to trust to plugging. Hamilton of Adelaide, where chronic alcoholism is more rife even than here, has made some interesting observations on this point; and Dr. Brown Kelly has explained the proclivity of the ethmoidal veins to bleed by their close connexion with the intracranial circulation. It is affirmed that in nose-bleeding due to enlarged liver, sinapisms, cupping or blistering to the right hypochondrium are efficacious. Inhalation of nitrite of amyl has also proved successful.

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G. M.

TUBERCULOSIS AND LUPUS

By GREVILLE MACDONALD, M.D.

For purposes of clinical convenience it is permissible to retain the old-fashioned distinction between tuberculosis and lupus of the nose.

Tuberculosis is generally described as occurring in the form of tumours of the cartilaginous septum, varying in size from a small granulation to such a bulk as would completely fill the nasal fossa. The surface is smooth or irregularly granular, covered or not with epithelium, and varying in colour from a grey to a dark purple. The tumours bleed easily on examination with the probe, the inner portions being harder than the periphery. After complete removal there is usually but small liability to recurrence; and though the wound may be slow in healing, cicatrisation is often rapid and firm. Ulceration may subsequently result, and sometimes is far more intractable than the tumours. The latter are sometimes indistinguishable from large fungating granulations, and may completely fill the nasal fossa of one or both sides, and even distend the nasal bones. Only exceptionally are the lesions found in other situations than the septum. The progress of the disease appears to be extremely slow as compared with the same affection in the pharynx or larynx, though, in one of my own cases, the tumour attained its full size in three months.

The *prognosis* is fairly satisfactory provided the patient is kept under constant observation, but the cicatrices are specially apt to break down. Yet it must be borne in mind that, at the worst, the advance of the nose affection is very slow, and that the patient may succumb to systemic infection before great destruction has taken place.

Treatment consists in the eradication of the growths and granulations, either with the sharp spoon or with the electric cautery. After careful cleansing and removal of crusts, the surfaces should be dusted with iodoform powder or painted with a fluid iodoform ointment and the nose finally plugged with cotton-wool to prevent the access of air, and thus the inspissation of the secretions. Beyond treatment on rational principles, it is doubtful how far any of the supposed local specifics for tuberculous ulceration are of any service; yet lactic acid in some cases proves invaluable, while tuberculin must be allowed a trial. In some cases, however, and probably more frequently than is realised, the disease may attack the mucous membrane of the nose, without any lesion of the skin in the immediate vicinity or elsewhere.

Lupus of the nose is always seen first on the anterior part of the septum. It appears, in the first place, as an infiltration of round, well-defined, flat, discrete tubercles, causing a general tumefaction. They may be paler than the normal colour, though they sometimes present a brownish tinge. But, whereas on the skin they are on a level with or slightly depressed below the surrounding surface, on the septum they usually appear slightly raised. Indeed, at first sight, it is not altogether easy to determine whether they are wide, flat, dry granulations, or tubercles covered with epithelium. Their surface may further be traversed by a few tortuous vessels. In the nose they are not so often in the form of minute, rounded prominences, closely packed together over large areas, as they usually are in lupus of the pharynx and larynx. Ultimately, on the septum, as elsewhere in mucous membranes, they coalesce, forming extensive raised patches with an uneven surface. The superficial epithelium becomes greatly thickened, and, mixed with mucus drying on the surface, it peels off in scales which may remain partly attached, sometimes overlapping one another in greenish-yellow scales. Deep ulcerations, sometimes fissure-like, may form in the midst of the infiltration, or the whole surface may break down into ulcers with a red, granular floor. Lastly, the tubercles may slowly shrink and disappear, leaving depressed shining cicatrices. Necrosis of bone does not occur, though the osseous septum as well as the cartilaginous may be absorbed. As the disease advances it slowly extends over the floor of the nose and ascends the outer wall. In this manner the turbinals may be destroyed, and the cartilages of the alae may be absorbed from the inside before any sign of the mischief has made its appearance externally.

Diagnosis.—The only disease which might be mistaken for lupus vulgaris is syphilis. The main point in distinction is the greater rapidity of advance in syphilis. Tubercle bacilli are seldom found in the scrapings, and even inoculations may prove negative.

The *prognosis* of lupus affecting the mucous membrane of the nose is entirely good, provided the patient understands that the treatment may extend over many years. It must not be forgotten that lupus may disappear spontaneously, to break out afresh or not.

The *treatment* of lupus of the nose demands considerable experience for its successful and safe conduct. Care is especially required in the prevention of scarring and unsightly cicatrices, both externally and within the fossae. Destructive treatment must be scrupulously limited to diseased regions. Of destructive agents the two most approved are the electric cautery, which was introduced for this purpose by Hebra, and the method of scraping or scarifying as advocated by Volkmann. Patient and systematic watching of the case and the curetting of each fresh appearance of granulation afford the only hope of cure. Yet so satisfactory is this plan that no other appeals to the experienced specialist. The use of tuberculin will, of course, be considered in some cases. Finsen treatment and that by radium are scarcely applicable to the nasal fossae, where surgery is far more successful than in lupus of the skin, and where disastrous scarring need not be feared.

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G. M.

SYPHILIS

By GREVILLE MACDONALD, M.D.

Primary syphilis of the nose is very rare, though cases have been recorded. The rhinologist has no advantage over the dermatologist in diagnosing a chancre in this region, as the point infected cannot be deeper than the vestibule.

In **secondary syphilis** also it is exceedingly rare to find any diagnostic signs of the disease in the nose, though it is asserted that occasionally mucous patches may be found on the septum, on the inferior turbinal at its anterior extremity, or on the inner aspects of the alae. More often, when there are numerous mucous patches in the pharynx, they may extend for a certain distance into the post-nasal space. But whether mucous patches be present or not, a catarrhal condition of the Schneiderian membrane is very frequent within the first few months after infection. The middle spongy body may assume a congested oedematous appearance, which subsides as the other symptoms disappear under mercury.

Tertiary syphilis of the nasal mucous membrane sometimes occurs very early. Cases are on record in which necrosis of the nasal bones has occurred as early as the seventh month after the primary infection, but usually the tertiary symptoms do not appear until after an interval of two to five years. The ulcerations rarely present the form, seen so often in the pharynx, which results from breaking down of a gumma. Usually the ulceration spreads slowly, leaving large suppurating surfaces or plates of necrosing bone, and giving rise to the horrible fetor which is so characteristic of the disease and always indicates the presence of necrosed bone. Anosmia supervenes, ulceration of the palate follows, and from subsequent perforation the voice becomes nasal. The nose falls in either (1) from destruction of the cartilages and the subsequent contraction of cicatrices; or (2) from the destruction of the osseous septum, whereby the support of the bridge of the nose is withdrawn. Most of the triangular cartilage may be destroyed without deformity, provided its anterior margins be intact; nor must it be forgotten that primary perichondritis and periostitis of the septum, leading to absorption of cartilage and bone without any fetid discharge or separation of sequestra, may lead to a falling in of the bridge of the nose. Traumatic abscess of the septum may also lead to cicatricial indrawing of the nose, even without perforation.

Examination of the interior of the nose may shew sharply defined ulcerated patches on the septum, and necrosed bone at the base of the ulceration, either loose or firmly attached. The whole of both turbinals may undergo molecular absorption without exfoliation, in which case the resulting condition, as well as the stench from the dried and putrefying secretion, may closely resemble that of atrophic rhinitis. Gummatous lesions of the nose are not common, and generally affect the septum, on one or both sides. They are reddish, sometimes purplish, though occasionally paler and soft; sometimes they give rise to pain. Occasionally fungating granulations are mistaken for gumma, and the surgeon is surprised that they do not respond to iodides. The surrounding ulceration and microscopical evidence that the excrescence is uncovered by mucous membrane or an underlying sequestrum will prevent error.

J. N. Mackenzie has drawn attention to a not uncommon form of syphilis which he calls syphilitic fibroid degeneration. The changes are met with in long-neglected cases, and especially when associated with alcoholism. While it is the septum which mostly suffers from necrosis, these changes affect by preference the turbinals. They are "very much enlarged, and present the appearance of dense, hard, whitish-yellow or red sessile masses, or are converted into distinctly pedunculated growths, which not only resemble, but are, in actual fact, true fibroid polypi of this region. . . . They are sometimes attacked by ulceration, and in this way partially destroyed, or they may be bound, as the result of the ulcerative process, to opposing structures by dense bands of cicatricial tissue."

The *diagnosis* is considered under the heading of Lupus (p. 29).

The *prognosis* of syphilis of the nose is always grave, because of exfoliation, and also because it is sometimes impossible to remove large sequestra. Before extensive necrosis has occurred, much good may be anticipated from constitutional treatment. In cases in which, from the presence of the characteristic odour, it is certain that there is exfoliated bone, and in which the discharge is flowing from the ethmoidal cells, the gravest fears must be entertained; for more or less of the ethmoid bone may be necrosed, which, from its position, cannot be removed either by natural processes or by surgical interference.

Treatment.—There is little to be said beyond what is conventional. Cleanliness and the iodides are essential, while it must not be forgotten that many cases of necrosis are even in this enlightened day caused by the abuse of mercury. Fungating granulations must be scraped or cauterised, and the ulcers efficiently dressed with iodoform. Necrosed and separated bone may be removed with impunity from the lower or middle passages; but when the dead mass lies in the superior meatus, the greatest caution must be exercised.

Inherited Syphilis.—It is not necessary to give more than a passing reference to inherited syphilis of the nose. In the earlier stages it is impossible to distinguish the syphilitic catarrh of infants from that of simple rhinitis. The persistence of a muco-purulent catarrh is sufficient to justify a suspicion of syphilis, while if the discharge become purulent and fetid the probability of the constitutional affection will appear great. Some writers have stated that hereditary syphilis may appear in the nose in older children, and that tertiary syphilides of the nose may occur at puberty as congenital lesions. Other authorities maintain that many cases of simple ozaena are due to the constitutional contamination. Nevertheless, the usual evidences of this in such cases are exceedingly rare, though I have seen two sisters suffering from ordinary atrophic rhinitis whose mother was suffering from tertiary syphilis of the nose. Further, a brother of these had lupus and destruction of the triangular cartilage, and a younger sister a troublesome muco-purulent rhinitis.

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G. M.

NEW GROWTHS OF THE NOSE

By GREVILLE MACDONALD, M.D.

Mucous Polypus.—The production of polypus is either dependent upon, or consists in a curious oedematous inflammation of the mucous membrane. The rule that this oedema, with the symptoms of paroxysmal sneezing and often asthma, so generally antedates by many months the development of the neoplasm, is often overlooked in discussing its etiology and classification. Sometimes, however, polypus is attended by suppuration, in which case the initial factor is probably ulceration. As the immediate consequence of such an accident we find granulation tissue covering the ulcerating surface. The longer cicatrisation is delayed the larger these granulations become; and, being perpetually bathed in mucus, they absorb moisture, become oedematous in fact, and are thus increasingly prone to fungate. This process is precisely that of an ordinary ulcerated surface where granulations are proliferating freely. If such an ulcer be bathed constantly in water the granulations become watery, pale, and flabby, and are scarcely to be distinguished histologically from many specimens of simple mucous polypus.

As this incipient polypus grows older its structure becomes modified, owing to the production of fibrous elements. The growth becomes more prominent and the blood-vessels more developed, especially towards the base, where the fibrous element grows firmer and contracts the surrounding tissue; thus a pedicle gradually is produced containing fully developed vessels which ramify in the peripheral and more oedematous tissue. Usually the structure ultimately becomes quite distinct from the surrounding surface from which it springs, although very often the mucous membrane in the immediate neighbourhood is in a condition of pronounced hyperplasia. As this granulation-polypus increases in size we find, curiously enough, that the epithelium tends to creep over it from the base; and it is in this way that these growths are often found completely covered with ciliated epithelium. Woakes taught many years ago that polypus is but a symptom of ethmoiditis.

Rindfleisch has described a fine reticular formation in the ordinary fungating granulation; and in the polypus we may find, associated with such reticulum, round cells which become larger and fusiform in various places, and are gradually converted into fibrous tissue. At an equal rate with the increase of reticulum the tendency to absorb and retain water becomes more pronounced. Amid this increasingly coarse reticulum we find, in different parts, various quantities of cells of diverse shape and size; varying, that is to say, from the small granulation to the long fusiform cell, which probably produces the fibrous element. Such I believe to be the essential structure of a mucous polypus. Formerly pathologists commonly described polypus of the nose as either myxoma or adenoma; yet for my part, although I have examined some hundreds of specimens, I have never succeeded in finding a true myxoma cell. In the younger growths we may find a cell apparently branched at the points where the fine reticular fibres cross one another, yet an unmistakable myxoma is never seen.

But although, clinically speaking, the initial inflammatory attack which results in polypus may be accompanied by suppuration, yet far more frequently the growths originate from a chronically oedematous and unbroken mucous membrane. A succession of acute attacks of cold in the head may gradually induce a chronic infiltration, weakening, and thickening of a certain area of mucous membrane; the fibrous elements becoming softened and granular, gradually disappear in the mucous fluid which steadily increases in the interspaces. New cells are produced which, accumulating in clusters, gradually pass through all the changes of organisation till a structure recognised as polypus is produced. But it must not be forgotten that in the production of this chronic oedema with its excessive sneezing there is a neurotic element, so that the tendency to sneeze, apart from cold-taking or direct irritation by foreign particles, appears to precede the oedema. This tendency, moreover, is distinctly hereditary, as is the asthma with which it is so often associated, as well as the typical hay-fever. This last, also, not uncommonly leads to the production of polypus. These changes are essentially in the muco-periosteum, so that the bone-tissue itself takes part; and a rarefying osteitis or osteophytic periostitis is frequently found in conjunction. So far we are justified in ascribing all polypoid degeneration to ethmoiditis. Recent authorities (St. Clair Thomson, Lack, Parker, and Luc) agree in this view, and probably it is now generally adopted.

Clinically the inflammatory origin of such growths is indisputable. For, as I have pointed out upon the authority of Rindfleisch, ordinary fungous granulations may contain a quantity of well-formed mucous tissue, presenting a pale pink, watery appearance, sometimes even yellow and jelly-like. Virchow, moreover, teaches us that mucin is a common product of irritated connective tissue, and that thus it must be admitted as a product of inflammation.

In cases in which the initial inflammatory attack is of sufficient intensity to produce molecular necrosis of the mucous membrane, in which, moreover, the tendency to heal is not strong enough even to produce a polypus, the ulceration may extend to the muco-periosteum and expose the subjacent bone. In such a way there may be masses of ordinary granulation-tissue, well-formed polypi, and carious bone coexisting side by side; and where such a process takes place within any of the accessory cavities we then find abscess and other consequences attendant upon the retention of pus.

Very frequently a portion of mucous membrane, especially when depending from the free border of the middle turbinal, presents such an appearance as makes one doubtful whether it should be considered as a diffuse polypus or rather as a mass of hyperplasia; nor will the microscope materially assist us in drawing the distinction. And if such a fragment do not appear sufficiently translucent to justify its being considered as polypus, we may, by soaking for a few minutes in water after removal, so increase its size as to give it microscopically every characteristic of ordinary polypus tissue.

So far it is quite intelligible how pathologists came to regard this oedematous inflammatory tissue as myxoma. But it is less easy to account for their designation as adenoma seeing that the growths for which the name polypus is now reserved by rhinologists never present any glandular structure, except, indeed, in cases in which the neoplasm consists rather of an oedematous hypertrophy of a widely attached piece of mucous membrane. The truth is that the surgeon has habitually confounded with polypus those curious lobulated or cauliflower-like growths which consist entirely in a hyperplasia of the mucous membrane covering the erectile tissue more especially developed over the inferior turbinals. In such growths the normal mucous glands are often largely increased in size and number, and one readily realises how the name adenoma was applied to them. But, according to usual observance, the discussion of these growths belongs rather to the domain of chronic hypertrophic rhinitis, although there is no good reason for discussing polypi as new growths if the former are not to be similarly considered. And both these causes of obstruction very frequently coexist in long-standing cases.

Clinical Aspects.—On several occasions I have actually watched the inception of a polypus in an attack of acute inflammation; that is to say, in cases in which I knew that no suspicion of such growths had previously existed. The patient is seized with more or less severe pain, generally referred to the supraorbital region; while the intensity of the swelling and the obstruction to breathing are altogether out of proportion to the degree of inflammation on the opposite side. The acuteness of the pain points to inflammatory tension in the muco-periosteum, or perhaps in an ethmoidal cell. After two or three days of such pain I have seen a polypus appear in the middle meatus, perfectly translucent, pale pink in colour, and sharply defined. But the more ordinary course is for the patient, after complaining of a constant succession of colds in the head, or chronic paroxysmal sneezing, to find that his nose is becoming persistently obstructed. He tells us he is always worse in damp weather; he walks and sleeps with his mouth open; his eyes become watery and bloodshot; and sometimes his nose widens across the bridge. Unable to obtain any satisfaction from blowing his nose he is perpetually wiping it; he loses his sense of smell and taste, and becomes a woe-begone object. In the earlier stages sneezing is often a prominent and very distressing symptom; but as the obstruction increases the mucous membrane becomes less sensitive to tactile stimulation, and the sneezing may disappear altogether. Most of these worst cases become chronic bronchitics with or without asthma. So that we must add the dilated right heart and many other allied symptoms to the train of mischief started by polypus. But even if they are spared the bronchial complications, they always become anaemic and dyspeptic and suffer much from headaches. Often the asthmatic and bronchitic troubles mask the nasal altogether. By some authorities, especially in the German and American schools (Hack, Bosworth, and others), such symptoms are supposed to result from reflex action originating in the nasal mucous membrane, and quite recently Dr. H. A. Francis has revived this explanation. But the asthma is by no means always cured by removing polypi; and it is probably more correct to consider the bronchitic and nasal conditions as several local manifestations of a chronic inflammatory process pervading the whole tract of respiratory mucous membrane. I am prepared to emphasise this view, knowing full well that the rectification of nasal abnormalities other than polypus often results in a most remarkable cure of asthmatic symptoms. It is noteworthy that in very rare instances the removal of a large polypus is followed for the first time by symptoms of asthma, though, in my own experience, complete eradication of the growths with all concomitant obstructive conditions speedily and permanently prevents the recurrence of the bronchial symptoms. It is impossible to give a sure prognosis in long-standing cases of asthma attended by polypus (4). (*Vide* p. 67.)

Treatment.—Various remedies have from time to time been suggested for the absorption of mucous polypus, but as a matter of fact they nearly all result in failure. The larger growths are best removed by the cold snare. When the disease is extensive, the only hope of cure lies in the complete removal of the middle turbinals. The electric cautery is greatly over-estimated as a destructive agent, and should be used only with great caution because of the possibility of inaugurating fresh inflammatory action. It is essential to remember that the cure of polypus consists as much in removing the inflammatory conditions that are responsible for it as in its surgery.

Benign Growths of the Nose other than Mucous Polypi.—Besides mucous polypi there are other growths which must at least be enumerated in this place. Those curious cauliflower developments so common in hypertrophic rhinitis, especially as it affects the erectile tissue and the inferior turbinals, really belong to the section of rhinitis. They consist of erectile tissue infiltrated with large masses of granulation-tissue, maintained by some authors to be lymphoid. In old-standing cases they grow more fibrous, and under certain conditions lose their ruddy hue, becoming oedematous and colourless; in this event the fibro-cellular elements become infiltrated with mucin and water, and the growths very closely resemble many instances of ordinary polypus. When finely lobulated and very substantial they have frequently been mistaken for papilloma, and recorded as such.

Osseous growths are rare; they usually come from the base of the sphenoid or the ethmoid and increase very slowly. They fall within the province of the surgeon rather than in that of the rhinologist.

Papilloma is actually very rare in the Schneiderian membrane; though occasionally small specimens are found, attached to the septum or in the vestibule, which have all the appearance of such growths as found on other distributions of mucous membrane. I have seen a few examples of well-developed papilloma covering the surface of a long-standing mucous polypus, when projecting between the alae and constantly exposed to the friction of the handkerchief. Such a development has no connexion whatever with any tendency in the parent growth to originate malignant disease.

Malignant Disease of the Nasal Cavities.—Malignant disease of the nose falls rather within the domain of the surgeon, and needs but little notice in these pages. According to Erichsen, the transmutation of mucous polypus into carcinoma is by no means rare. That this is an error is an opinion which I believe every author will now accept. After having treated more than fourteen hundred cases of polypus, I have never seen these benign growths become malignant, although I have seen sarcoma and mucous polypus associated in the same nasal fossa. Sarcoma is decidedly commoner in the nose than carcinoma; and I may add, as a characteristic of the former in this region, that its malignancy is probably more difficult to estimate on microscopical examination than in other regions. Cases have been recorded of complete cure of sarcoma by intranasal operation alone. I watched a patient of my own who presented every symptom of malignancy, both clinical and microscopical, in whose case the surgeons declined to interfere, yet for over four years he presented no indications of recurrence. The treatment was entirely intranasal, and extended over three years; the tendency to recurrence, at first extraordinarily rapid, gradually diminished until it has ceased now, I believe, altogether. Probably all the cases of so-called fibrous tumour found in adolescents, which make such frightful local ravages, separating and protruding the eyes, flattening the nose, and producing "frog-face," are really sarcoma. Enchondroma has also been described as a cause of like symptoms.

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G. M.

MALFORMATIONS AND DISEASES OF THE SEPTUM

By ERNEST WAGGETT, M.B.

Anatomy.—Before discussing the diseases of the nasal septum, its anatomy must be described. In early embryonic life the rudiment of the septum can be recognised in the mesial nasal process dividing the two pockets of olfactory epithelium which make their appearance below the fore-brain. The two globular extremities of this mesial nasal process ultimately form the philtrum and adjacent portions of the upper lip, the intermaxillary bone, and also the portion of the nasal septum anterior to the naso-palatine canal, which marks the posterior boundary of the intermaxillary bone. In hare-lip the natural union has failed to take place between the mesial and either one or both lateral processes, and there is no difficulty in recognising in double hare-lip a definite instance of malformation concerning the nasal septum. The major part of the septum is formed from a mesoblastic mass projecting from the basisphenoid region and continuous anteriorly with the mesial nasal process. This fuses below with the palatine processes of the superior maxillary mass, the fusion failing to occur in the condition known as cleft palate. In certain cases of cleft palate no structure corresponding to the vomer can be found. A sheet of cartilage appears in the primitive septum during early fetal life. This cartilage is seen, in vertical sections, to end above as the crista galli and below as a rounded edge which is embraced by the lips of a bony groove. This groove is the upper edge of the vomer, which ossifies from connective tissue and not from cartilage. In the fetus a capsule of cartilage and a sac lined with specialised epithelium can be seen on each side of the vomer. This is Jacobson's organ, which in many animals receives branches of the olfactory nerve, and is a well-developed sense organ, but in adult man this structure has rudimentary proportions only.

In the adult the nasal septum consists of three distinct portions. An oblique line running from the rostrum of the sphenoid behind to the anterior nasal spine in front forms the upper boundary of a plough-shaped lower portion. This, the vomer, articulates posteriorly with the under surface of the sphenoid by two wing-like expansions, and below and in front with the hard palate and anterior nasal spine. Its long upper edge articulates posteriorly with the perpendicular plate of the ethmoid, and in front it is more or less deeply grooved for the reception of the edge of the quadrilateral cartilage. Its posterior free edge separates the two choanae. The remainder of the septum is at one time wholly cartilaginous, but the posterior portion is, during early life, invaded by ossification from above. The result of this ossification is the formation of the so-called perpendicular plate of the ethmoid; the size of this plate and the proportion which it bears to the unossified remainder vary considerably. As a general rule the bone extends forwards to articulate with the under surface of the nasal bones, reaching to the junction of the lower with the middle third of the latter. Union with the upper edge of the vomer occurs about the fifth year and extends rather in front of the central point of the bone. Between the two, a slip of cartilage remains unossified. This, the processus sphenoidalis, is more or less completely encased in a bony tube formed by the two bones. The remaining unossified portion of the septum is known as the quadrilateral cartilage of the septum. Its anterior superior border articulates with the nasal bones and the triangular or lateral cartilages of the nose, while its antero-inferior border is free and may be felt as a convex edge among the softer structures of the columella. This anterior corner of the septal cartilage serves the important function of supporting the alar cartilages. These structures provide the nose with its permanently patent orifices set in a horizontal plane. Posteriorly the cartilage articulates with the perpendicular plate, while its lower edge and the processus sphenoidalis, which is continuous with it, are contained within the groove upon the upper edge of the vomer. The mucous membrane covering the septum is intimately blended with the underlying perichondrium and periosteum, which may be easily elevated from the hard structures, except in the region of the vestibule, where the cartilage and perichondrium are very closely united. A patch of mucous membrane containing numerous glands occupies an area the size of a threepenny piece, opposite the anterior ends of the middle turbinals. This thick portion of the mucous membrane, together with an underlying thickening of the quadrilateral cartilage, constitutes the tuberculum septi. Occasionally in the posterior region, the mucous membrane is thrown into raised, parallel folds which run in a more or less horizontal direction; these folds contain lymphoid tissue and may appear in the post-rhinoscopic picture as pale elevated cushions. The ciliated columnar epithelium covering the general surface of the septum changes anteriorly into the epidermis of the vestibule, whilst an area of olfactory epithelium is found in the posterior superior region. The main arterial supply is from the naso-palatine branch of the spheno-palatine artery; this reaches the septum after crossing the sphenoid, and divides to supply the ethmoidal and the vomerine regions of the septum. A branch of the lower division traverses the anterior palatine canal; there is also an anastomosis with branches from the superior coronary artery. In the anterior region of the septum above the intermaxillary bone the mucous membrane is thinner than elsewhere and contains a network of large capillaries; from this, the so-called area of Kiesselbach, haemorrhage very commonly takes place, either spontaneously or as the result of injury.

IRREGULARITIES.—The septum claims attention mainly on account of the irregularities in its form which cause nasal obstruction. These irregularities may be considered as falling into three classes, though often enough two or three of them are found in a single case. These three classes are: (1) developmental deflections; (2) crests and spurs; (3) traumatic deformities.

Developmental Deflections.—In young children the nasal septum is, as a rule, almost straight, and this also holds good in the adults of those races whose physiognomy does not widely depart from the infantile form; but in the long and narrow type of face commonly seen in this country it is quite unusual to find a perfectly straight septum. The deformity is clearly due to a lack of uniformity in the growth of the various elements of the facial skeleton. Deflections of this class are best studied in the pathological condition known as the adenoid facies (*vide* p. 94).

During the period of the second dentition, between the ages of six and thirteen years, the facial skeleton undergoes very rapid growth, losing its infantile character and attaining the adult form. Where adenoids exist, the mouth is held open for purposes of respiration during a great part of the twenty-four hours; and in consequence of this, one of the most powerful of the moulding factors, namely, the constant lateral pressure of tongue against the alveolar arch, is withdrawn, and the well-known narrow, gothic-arched palate results. The upper portion of the vomer continuing to grow longitudinally, finds in this form of palate an unduly resistant obstacle, and is compelled to assume a curved instead of a rectilinear form, being as it were mastered by its surroundings instead of taking part in their moulding. As a rule, the upper edge of the vomer in the cramped nose of the adult adenoid facies is S-shaped, and the quadrilateral cartilage and perpendicular plate are necessarily also thrown out of the straight, so that in vertical as well as in horizontal sections they present a curved figure. Thus it often comes about that while one nostril is obstructed anteriorly by the convexity of the quadrilateral cartilage and anterior part of the vomer, the other is obstructed near its posterior end by the convexity of the perpendicular plate and posterior part of the upper edge of the vomer. It is only the rapidly growing portion of the vomer, namely, the upper edge, which becomes distorted, the postero-inferior portion always remaining in the middle line; notable inequality in the size of the two choanae is exceedingly rare.

The gothic-arched palate is certainly not the only cause of septal deflections of this type, for they are not infrequent in persons whose facial skeleton is in other respects well formed. In all cases, however, they seem to be associated with the extremely rapid development of the vomer during the later years of childhood, and are not seen before the age of six years. There is also no doubt that many cases of notable deformity have resulted from abnormal growth occurring in a septum which has been thrown slightly out of the straight by mechanical influences, such as falls upon the face in childhood, and possibly the constant twisting of the part by the use of the handkerchief or the habitual pressing of the feature into the pillow at night. A very conspicuous type of deformity is often seen in which no suspicion of an adenoid origin can be entertained, and in which the nose is broad and otherwise well formed; here the defection is C-shaped, one nostril being unduly patent throughout at the expense of the other. A groove often one-third of an inch deep is seen upon the concave side corresponding with the upper edge of the vomer and with a prominent ridge upon the convex side. This condition is clearly not traumatic, as the deformity concerns the middle rather than the anterior portion of the nose. Mosher points out that in many cases there occurs in association with delayed eruption of the incisor teeth of one side, an inequality in the development of certain bony ridges, the wings of the intermaxillary bone, which practically form an anterior continuation of the lips of the vomerine groove. The unequal development and abnormal cant of these ridges serve to tilt the edge of the growing cartilaginous septum over to one side, and thus to produce developmental deflection of the whole septum.

A form of deflection of a somewhat different character, and chiefly affecting the perpendicular plate, occurs in adult life as the result of pressure by an enlarged middle turbinal bone. In the same category may be placed deflection due to the presence of large polypi or new growths.

Crests and Spurs.—Irregularities in the thickness of the septum apart from those due to fracture are quite uncommon. On the other hand, in at least one out of every three adult noses, examined in routine practice, a very definite shelf or crest is seen running obliquely upwards and backwards from the neighbourhood of the anterior nasal spine. This ridge follows, in fact, the line of the upper edge of the vomer, and may be termed the vomerine or lateral crest. It may occur on both sides of the same septum, but in that event the crest on one side will be found in the anterior portion only of the nose. These crests may extend backwards along the vomerine line as far as the articulation with the sphenoid. As a general rule they increase in width or prominence as they pass back, and often culminate in a definite pointed eminence in the posterior vomerine region. Such an eminence or spur, as a rule, projects between the posterior portions of the middle and inferior turbinals, and may be sufficiently prominent to touch or even pierce the antro-nasal wall. When removed, a well-developed crest is found to consist of cartilage often supported by a lip of bone; the spur, on the other hand, consists either of solid bone or of cartilage more or less completely encased in a shell of bone. The formation of these structures appears to be due to the growth of the edge of the cartilage which has escaped from the vomerine groove on account of the deficiency of one or both of its bony lips. In the same way the posterior portion of the ridge and the spur are due to overgrowth of the processus sphenoidalis of the cartilage, the bony casing of which is often defective or very thin upon one side.

Although these lateral crests and spurs are exceedingly common they are of no clinical importance in the vast majority of cases. Indeed it may almost be said that they hardly ever call for surgical consideration except when they are associated with a deflection of the septum. This combination is quite common, and when it occurs the crest is always upon the convex side of the distorted structure. Occasionally large crests upon a straight septum give rise to trouble and need interference, because they cause either pressure symptoms or nasal obstruction. The edge of a crest may come into apposition with the inferior turbinal in its anterior half; this produces a *cul-de-sac*, closed in front, from which secretions cannot be expelled by expiratory efforts. The patient complains of the passage of a muco-purulent discharge into the back of the throat, and thus one of the forms of so-called "post-nasal catarrh" is brought about. Cases of this kind of nasal obstruction may easily be overlooked by those unused to the examination of the nose, for the organ may appear perfectly normal above the level of the crest. Those who are not constantly engaged in rhinological work will find it a fairly safe rule, when any suspicion of abnormality exists, to produce shrinking of the tissues with weak cocaine or adrenalin during examination of the nose. If this is done very prominent spurs will often be found far back in the nose, the presence of which would never have been suspected from a cursory examination. Repeated experiences of this kind will, moreover, convince the observer that in the great majority of cases these structures cause no symptoms whatever, and will thus prevent him from making the mistake of performing a number of small surgical operations from which his patient will derive no benefit.

Traumatic Deformities, Fractures, and Dislocations.—Fractures of the septum concern chiefly the quadrilateral cartilage, in a much less important degree the perpendicular plate, and practically never the vomer. Traumatic deformity is quite common in this country, and generally results either from boxing or from falls upon the face, such as occur in hunting and on the football ground. After really severe punishment the nose may be hammered into the face so as to produce the characteristic pug-nose of a prize-fighter, but far more often the tip of the organ is twisted towards one or other cheek. Probably the commonest fracture of the kind is that caused by a heavy blow from the opponent's right fist in boxing. The tip of the organ is then deflected towards the right cheek, and it is important to notice that the nasal bones are definitely displaced, that on the left being flattened towards the plane of the face, while that on the right approaches the plane perpendicular to it: the slope of the left nasal bone is shallow and that of the right steepened. Such a deformity is diagnostic of trauma. Careful examination of a typical case will shew that the left nasal bone has been fractured through a line dividing the lower from the middle third, that is to say, the lower portion which projects beyond the articulations of the perpendicular plate has given way. The right nasal bone has been dislocated into a more or less sagittal plane and the anterior part of the perpendicular plate may be just cracked, but it is difficult to detect definite signs of fracture. The quadrilateral cartilage has been fractured along a vertical line a little internal to the pyriform opening (the bony orifice of the nose), and the anterior fragment has been driven into the nose, and also rotated round a vertical axis. These two phenomena together decrease the prominence of the feature and carry its tip towards the right cheek.

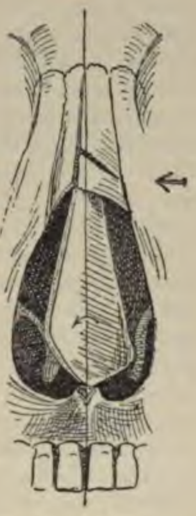

FIG. 1.—A common form of fracture in boxers. The nasal bone is fractured in its lower third, and the quadrilateral cartilage is fractured vertically just within the pyriform opening. The anterior fragment is driven into the nose and the tip of the organ is rotated towards the right cheek. [From the Author's *Diseases of the Nose*.]

There are many variants of this form of fracture; not uncommonly the cartilage is broken along a line parallel to the edge of the vomer, with or without the association of a vertical fracture. It is also common for the lower edge of the cartilage to be dislocated from the vomerine groove. When this occurs during the years of growth, the deformity becomes increasingly marked as time goes on. Dislocations of the cartilage also produce a marked deflection of the edge of the part into one or the other vestibule. When this condition is present the vestibule is found to be obstructed by the rounded smooth edge of the septum, in front of which the displaced inner limb of the alar cartilage may be seen and felt. This appearance in the vestibule is, however, not always due to traumatic dislocation but sometimes definitely depends upon developmental distortion.

The differential diagnosis may be gathered from the description of the three categories of septal deformity. In many cases it is impossible to determine for certain whether a deflection is purely developmental or purely traumatic; this is because the developmental deflections are very common and receive their full share of violence, while on the other hand a deformity due to fracture and dislocation occurring in childhood invariably interferes with the subsequent growth of the septum, which consequently takes on some of the characters of a developmental deflection. It may, however, be stated generally that large sweeping curvatures, and especially the S-shaped deflections, are due to irregularities in growth, while abrupt irregularities as seen in the horizontal section are always due to injury. In both cases an acute angle may be seen in vertical section along the line of the upper border of the vomer, and in no class is such an angle more acute than in the developmental deflection combined with the presence of a spur on the convex and a deep groove on the concave side. Deviation of the tip of the feature may occur in both classes, but when such a deformity is associated with extreme asymmetry in the region of the nasal bones, the condition is always traumatic in origin. When a case is seen immediately after an injury, the diagnosis of traumatic deflection must largely depend on the assertions of the patient or his friends as to the previous symmetry of his features. If the nasal bone region is deformed, and especially if crepitus can be detected, there will be no difficulty; but when the patient merely complains of a nasal obstruction, whether unilateral or bilateral, which has quickly followed the receipt of the injury, the practitioner must not immediately jump to the conclusion that the septum has been fractured. The obstruction may prove to be due to haematoma or even to inflammatory swelling superadded to a previous deflection.

The great majority of persons with deflected septums and spurs have no symptoms, and these physical conditions are only discovered during the routine of examination. Symptoms when they exist fall under the heads of: (1) intranasal pressure, (2) nasal obstruction, and (3) undue patency. These subjects will be dealt with briefly here.

Symptoms of intranasal pressure only occur when a deformed septum comes into a close apposition with structures upon the outer wall of the nasal fossae, so that the least turgescence of the mucous membrane is able to bring pressure to bear upon the nerve endings. Approximation of a lateral crest to an inferior turbinal, large enough to cause marked or even complete unilateral obstruction, may not be so close as to produce pressure symptoms. It may be pointed out in passing that though these cases of septal pressure are common, the deformities are so slow in development that ulceration of the mucous membrane is never seen, though the pressure exerted may be sufficiently great to cause absorption, or at all events defective development of the turbinals. Pressure between lateral crests and the inferior turbinal is held to be responsible for referred pains in the region of the upper jaw and in the ear, but experience shews that the connexion can be clearly proved in only a very small proportion of cases. It is quite the reverse in cases in which the anterior end of the middle turbinal presses against the septum. Such pressure may occur without any septal deformity, but it is commonly seen when enlargement of the middle turbinal gradually causes deflection of the perpendicular plate. When dealing with such cases it must be remembered that the septum may be thrust over until it presses upon the opposite middle turbinal, and that relief may not be obtained if the larger middle turbinal only is reduced by operation. Pressure in the region of the middle turbinal causes a sense of weight and even severe pain across the bridge of the nose, and during exacerbations which occur in all these cases when the tissues are specially engorged in acute catarrh, constipation, or the catamenia, there will be severe pain and tenderness over the points of exit of the infra- and supra-orbital nerves of the corresponding side. Tenderness in these positions is diagnostic of pressure between the middle turbinal and the septum, and not of sinus suppuration, though the latter may be a contributory cause by inducing hyperaemia or hyperplasia of the mucous membrane of the middle turbinal. These nasal pains may be referred to the eye as well as to the vertex and other regions of the head, and there is no doubt that a considerable proportion of patients suffering from periodical attacks of severe hemicrania are entirely relieved by the removal of the anterior ends of middle turbinals pressing upon the septum in its most sensitive region.

Nasal Obstruction.—It is only necessary to state here that the septum is an important factor in the causation of the great majority of examples of obstruction in adults. With the exception of cases of polypi and hypertrophy of the mucous membrane of the turbinals, permanent and well-marked nasal obstruction which is not due to injury is nearly always associated with mal-development of the nasal fossae. In such cases of cramped nose the septum is, with the rarest exceptions, deformed, and it is by operative interference with that structure that relief is generally best obtained.

The obstruction due to septal deflection or crests can scarcely be said to present any characteristic train of symptoms. The following condition is, however, noteworthy; a patient frequently complains of bilateral obstruction, and yet his nose, on cursory examination, appears to be blocked on one side only by a deflected cartilage. A more searching scrutiny after shrinkage of the tissues with adrenalin and cocaine will probably reveal an S-shaped deflection, the posterior limb of the S blocking the opposite nostril in its posterior third.

Undue Patency.—In a comparatively small number of cases of marked C-shaped deflection, one nostril may be much blocked and the other unduly roomy. In most cases of septal deformity the concavity is more or less filled up by overgrowth of the corresponding inferior turbinal. In the exceptional cases referred to, either a condition of rhinitis sicca or one indistinguishable from ordinary ozaena may exist (cf. p. 21).

Treatment.—It is impossible to insist too often that deformities of the septum as such require no treatment except in the rare instances in which there is serious disfigurement without any other manifestations. Treatment is only undertaken in order to remove the symptoms mentioned above, and generally it is nasal obstruction and its effects upon the respiratory apparatus and the ears that bring these cases into serious consideration. It is said that the removal of spurs may be required to facilitate the passage of Eustachian catheters, but I have never found this necessary; for though a crest is in close apposition with the inferior turbinal anteriorly, a considerable amount of room will be found below it, when once the narrow spot is passed by the tip of the catheter held in an inverted position.

In dealing with nasal obstructions the general rule is that if some structure must be removed it is the septal deformity and not the valuable mucous membrane of the turbinals that should be sacrificed. It is perhaps necessary to add that in the numerous cases of obstruction due to nocturnal engorgement of the cavernous tissue of the inferior turbinals, which swell and leave an impress visible in the morning upon the surface of the septum, no tissue at all need be removed. Such cases will be relieved by a course of strychnine and iron with aperient pills, or at the worst will require a scarring of the turbinal mucous membrane with a few strokes of the galvano-cautery. When this condition of vasomotor obstruction coexists, spurs and deflections should be left alone until the mild treatment just mentioned has had a proper chance of success. The galvano-cautery should not be employed to reduce septal deformities; the same may be said of electrolysis, which has proved to be both tedious and painful.

Operations upon Crests and Spurs.—The removal of crests and spurs from a comparatively straight septum is not often necessary, unless they are of considerable size. When small they may be removed very speedily by the electric trephine, or, in the case of cartilaginous crests, by the spoke-shave of Carmalt Jones. Both these methods generally leave a surface denuded of mucous membrane, which means delay in healing and crust-formation. A neater result is obtained with the saw; an operation, not necessarily occupying more than sixty seconds, leaves the patient with a linear wound which heals in a day or two. The relative advantages of local or general anaesthesia need not be discussed here; practically all the operations on the septum can be performed without severe pain under cocaine, but it is often a question whether the nervous and emotional strain does not exhaust the patient more than a well-administered general anaesthetic.

Nitrous oxide is sufficient for many of the smaller operations if the surgeon is expert, but it should never be employed by those unused to these operations, for intranasal interference, unless it is done properly, is almost certain to do harm, giving rise to adhesions and to cicatrices in the mucous membrane, which hamper the performance of subsequent operations. As a rule, it will therefore be wiser to employ very light chloroform anaesthesia, in which the cough and swallowing reflexes are fully maintained. There is no doubt that all intranasal work is very much facilitated when the patient is in a sitting position, and the surgeon will do well to advise the anaesthetist to consult recent works (by Hewitt and by De' Prenderville) upon the administration of chloroform in the sitting posture. When a crest has to be removed under general anaesthesia the nasal vestibule is first cleansed and rubbed with $\frac{1}{1000}$ solution of biniodide of mercury in spirit. A strip of gauze soaked in $\frac{1}{1000}$ solution of adrenalin chloride is laid carefully over the part, and the inferior turbinal is painted with the same drug if its turgescence interferes with the view. When no general anaesthetic is given, 5 per cent solution of cocaine is brushed upon the crest, the inferior and middle turbinals, and upon the opposite side of the septum; that is to say, upon all parts which may be touched by the instruments. The septum is then papered with gauze soaked in adrenalin, and after a few minutes the parts will be sufficiently anaesthetic to receive a submucous injection of the local anaesthetic, and sufficiently anaemic to prevent its rapid absorption with the production of toxic symptoms. Either eucaine or cocaine will serve well for injection, a very convenient form being Braun's dental tablet (gr. $\frac{1}{6}$ of cocaine, with a little suprarenin and salt), dissolved in 2 or 3 c.c. of boiled water. A strong syringe with a specially large and long hypodermic needle is used, and the point of the latter is driven right down to the cartilage. If this is done there is no difficulty in injecting the fluid, which raises the muco-perichondrium in the form of a white weal. If the point be not carried deep enough the compressed fluid will immediately burst through the delicate mucous membrane and escape. Discretion must be used as to the number of punctures (one or two will suffice for a crest) and the dose of cocaine. If any doubt exist as to the patient's idiosyncrasy with regard to cocaine, it is advisable to give a prophylactic dose of sal volatile, and to avoid alcohol, as it favours haemorrhage. In ten minutes' time the septum will be perfectly anaesthetic (even with gr. $\frac{1}{12}$ of cocaine, if injected with a sufficient quantity of fluid), but the patient will, of course, feel the vibrations and mechanical shocks inseparable from any operation upon bone. The only special instrument required is a nasal saw; Goldsmith's pattern is very suitable, as it can neither "bind" nor bend. A narrow flat saw may embarrass an operator unused to it, by becoming firmly wedged in the bone, and, if unduly flexible, it may bend and leave an important mass of bone behind at the posterior end of the wound. The other instruments required are: a Thudichum's speculum, a strong tenotome, a narrow elevator, such as the flat handle of a probe, a pair of small sinus-forceps, or angled spring-forceps. It need hardly be added that whatever form of anaesthesia is employed, the work must be done with a forehead mirror and adequate illumination.

An incision, carried right through the muco-periosteum and perichondrium, is made along the free edge of the excrescence, commencing at the posterior extremity, or, more strictly, at the apex of the spur. The soft parts are then quickly elevated and turned upwards from the upper surface of the shelf, that below it being left alone. The saw, with its flat side against the septum, is then introduced either above or below according to the convenience of access, and the cartilage and bone are cut through with a few rapid strokes, care being taken not to injure the sphenoid with the tip of the instrument. The detached part, namely, the cartilage, bone, and the mucous membrane of the under surface, is then removed with forceps, and the upturned flap is plastered down over the wound, which it will be found to cover completely. The patient may be sent home, with instructions to recline quietly on a sofa, with the head raised, for some hours, to avoid exertion, excitement, and alcohol. Some haemorrhage may be expected about thirty to sixty minutes after the operation, when the reactionary hyperaemia which follows the adrenalin ischaemia sets in. No packing is employed, but every two hours (when awake) during the first forty-eight hours ten minims of ten-volumes hydrogen peroxide should be run into the nostril from a dropper (for example, the filler of a stylographic pen). The drug acts both as an antiseptic and as a powerful haemostatic, and should be used freely and often if troublesome haemorrhage sets in. I am strongly of opinion that packing should be avoided, and that the simple method just described should be employed as a matter of routine; for since adopting it, about five years ago, I have only once had occasion to pack a nose for post-operative haemorrhage, and have only twice observed fever indicating post-operative infection. A wound made and treated in this manner will appear to be perfectly healed in forty-eight hours, nothing abnormal being visible except the swollen condition of the flap. As a matter of fact a small raw surface is present far back where the mucous membrane on the posterior aspect of the spur has been cut away; consequently the patient should wear a piece of wool in the vestibule for a day or two if the atmosphere is dusty. The patient should never introduce his finger into the nostril. Although these operations are of very small moment, attention to detail in their performance on two or three occasions will prove very valuable as an introduction to nasal surgery. No mention has been made of any attempt to create an aseptic operation field, for, although living micro-organisms can be collected from the anterior regions of the nose, experience shews that these wounds invariably heal by first intention and without evident septic absorption. When the vitality of the mucous membrane is impaired by strong antiseptics this is not the case, and the use of the galvano-cautery, in conjunction with a cutting operation, should be especially avoided. Gauze packing also is apt to become septic if left in the nose many hours, and if it must be employed to arrest haemorrhage, peroxide of hydrogen should be injected as far into its interstices as possible, every two or three hours. Necrosis of bone or cartilage never occurs if these structures are covered with flaps of mucous membrane.

Operations for Deflected Septum. — Developmental and traumatic deviations may be dealt with under one head.

Quite recent fractures require separate consideration. It is best to deal with these cases immediately after the receipt of the injury; but if they are seen on the second or third day, when the parts are in a state of inflammatory oedema, it is well to allow them to settle down for a week under the influence of cold applications and rest. In characteristic cases of fracture-dislocation of the nasal bones, nitrous oxide should be administered, and the parts replaced with Walsham's or Adams's forceps. The nasal bone on the flattened side of the feature is first grasped between the two blades of the instrument (the outer blade being covered with rubber tubing to prevent bruising of the skin), and wrenched into position; the septum and the other nasal bone can then be brought into place with the same instrument. The nasal bones do not need any support unless the damage is excessive, but the septum may cause some trouble by the overriding of the fragments. In this event it should be kept in the middle line by introducing into each nostril a Lake's rubber splint or a hollow metal splint made on the same pattern but permitting nasal respiration. These must be worn for three weeks, and need not be removed before that time if peroxide of hydrogen is injected two or three times a day; this should be employed as a precautionary measure whether splints are used or not, for the mucous membrane is generally torn and the fracture is thus a compound one. External splints are unsatisfactory and scarcely ever required. For a day or two cold wet applications should be kept on the nose.

In cases of long-standing deflection, the inferior turbinal on the concave side is generally unduly large, and must be reduced by punching at the time of operation.

There are several operative methods for rectifying deflection of the septum; they are all useful in certain cases, but since the introduction of adrenalin a careful submucous resection of the distorted parts has become possible, and the other methods have been largely superseded. A description will be given of two of the latter operations, which can be performed rapidly, and are therefore suitable for cases of emergency.

Moure's Operation.—When the quadrilateral cartilage is mainly at fault, it may be brought into position by making two incisions about one and a half inch long and parallel respectively to the upper edge of the vomer and the bridge of the nose. These incisions are conveniently made with the button-hole scissors devised by Moure, and they are carried through both layers of mucous membrane and cartilage. The resiliency of the deformed part is in a measure destroyed by these incisions, and the convex portion can easily be thrust to and beyond the middle line. It must be kept in its new position by the insertion of a splint, either Moure's hollow metal splint or one of those previously mentioned. In either case the splint must be worn for three weeks—that is to say, until firm union in the new position has taken place. The immediate result in many cases is good, but the deformity is apt to return gradually, especially in growing patients.

Gleason's Operation.—When a single C-shaped curvature causes obstruction, and especially when there is an unduly roomy nostril on the concave side, a good result may be obtained in a few seconds by merely introducing a narrow saw below the convexity, and rapidly cutting upwards for about an inch. The semi-detached fragment now hangs, as it were, from a hinge above, and should be thrust with the finger through into the opposite nostril. Its lower edge will catch upon the lip of the perforation which has been made, and, therefore, as a rule does not need any splint to keep it in position. Healing generally takes place without leaving a perforation of notable size.

Although it is generally considered obligatory to leave a septum without any perforation, such a defect is, as a matter of fact, of no importance unless it be situated within the anterior inch measured from the limen of the vestibule. In this area a traumatic perforation is apt to cause discomfort by the accumulation of dried mucus and dust upon its edges, but in the posterior region this trouble does not occur, and in some cases a hollow convexity can be safely sawn off without any risk of permanent discomfort.

Submucous Resection.—We are indebted to Freer and to Killian for the elaboration of a definite method of shelling out all the distorted portions of cartilage and bone, leaving a perfectly flat septum composed of the two layers of mucous membrane, or, more strictly, muco-perichondrium and muco-periosteum. Since this method has been generally adopted, the treatment of nasal obstruction has been much more satisfactory than formerly. The exact details are still subject to alteration and improvement, but the instructions given below will, if followed, permit a tolerably dexterous operator to obtain a good result in a developmental deflection in about fifteen minutes. The rather longer duration of the operation, as compared with the rapid measures previously described, is counterbalanced by the considerations that the nose is perfectly healed within twenty-four or forty-eight hours, that no after-treatment is necessary, and, more important still, that unless some unfortunate accident occurs, the patient is certain to have a septum which will remain permanently straight, and that as far as his obstruction is due to the septum, he will remain quite free from its recurrence. There is no loss of mucous membrane, and, therefore, no perforation and no healing of a wound under a crust. Moreover, in the common S-shaped developmental deflection, it is the only single operation (with the exception of a somewhat rough method in which all the parts are comminuted) whereby both the anterior and posterior obstructions are removed.

The septum is anaesthetised and rendered bloodless by the measures described for the lateral-crest operation; either general or local anaesthesia may be employed. The greater part of the operation can be performed quite painlessly under cocaine, but where much deformity of the vomer exists, it is better to employ chloroform, as the fairly vigorous use of the mallet and chisel which is necessary sometimes entails rather severe nervous strain. The instruments required are: a tenotome; two small muco-periosteal elevators, slightly curved, one blunt, one almost sharp at the edge; Thudichum's speculum with long narrow blades (St. Clair Thomson's pattern is self-retaining and much more handy than Killian's median rhinoscopy speculum); Ballenger's swivel knife; small gouge with square or fish-tailed end; mallet; Adams's or Walsham's septum forceps; small nasal punch-forceps (*e.g.* Hartmann's conchotome, smallest size); rubber sheet for splints. The deformed parts are to be removed through a linear wound in the mucous membrane of the side which is convex anteriorly, or if the surgeon be not ambidextrous, through that of the left nostril. No distorted parts must be left remaining in front of this incision, which must, therefore, occasionally be made over the actual anterior edge of the quadrilateral cartilage. The muco-perichondrium is very adherent here, and requires careful dissection off the cartilage; whenever possible, therefore, the incision is made farther back, where elevation of the soft tissue will be found to be quite easy. The more or less vertical incision is about ¾-inch in length, and is to be carried at one stroke through the left (for example) muco-perichondrium and cartilage. The depth of the incision is best controlled by ocular inspection of the right or intact side. The point of the knife will be carried through the cartilage until it commences to pucker the mucous membrane; the downward course of the blade can then be easily controlled and directed by watching this pucker or elevation as it travels along the mucous membrane. The little finger or a wad of wool is then thrust into the right nostril until the cut edge of the cartilage shews up white and clear in the left nostril. With the sharp elevator the muco-perichondrium is very carefully defined and raised from the cartilage. If this is not properly done at the outset, an hour or more will very probably be occupied in laboriously splitting the muco-perichondrium into a superficial and

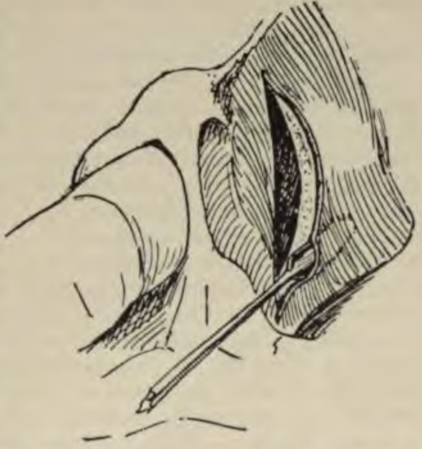

FIG. 2.—Submucous resection of the septum. Incision from the left side carried through the mucous membrane of that side and the cartilage. The cut edge of the cartilage has been thrust into the left nostril with the finger, and the mucous membrane is easily elevated from it. [From the Author's *Diseases of the Nose.*]

a deep layer, with frequent tearing of the former, and, at the best, an imperfect result. On the other hand, when this initial elevation is properly done, the remainder of the perichondrium can, in a developmental case, be elevated with the blunt instrument in a few seconds, and absolutely without any haemorrhage. In fracture cases in which fragments are overlapping and the perichondrial layers united by cicatricial fibrous tissue, a fresh careful dissection must be made as soon as the edge of the posterior fragment comes into the operation field. The blades of the long speculum are now introduced to embrace the denuded cartilage, which is cut away with one sweep of the swivel knife. A slip of cartilage, ⅓ inch broad, should always be left under the bridge of the nose to preserve the shape of the feature. In a developmental case the operation up to this point has occupied about five minutes, and has been quite painless, and it is reasonable to consider that it may well be substituted for the slightly more rapid operations previously mentioned. It should here be definitely stated that in cases of bad fracture deformity, considerable difficulty may be met with, and an hour may be spent in disentangling the fragments of the cartilage. These difficulties, however, are greatly diminished if proper ischaemia be induced, and if the exact conformation of the fragments be made out before the operation is begun.

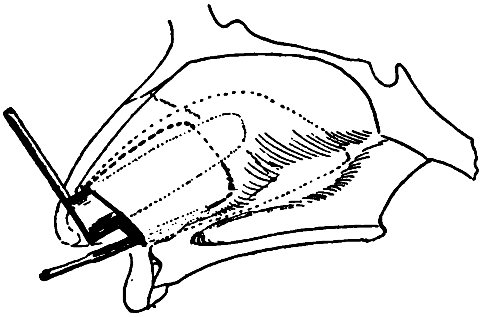

FIG. 3.—The fine-dotted line indicates the area of mucous membrane elevated. The cartilage, embraced between the limbs of St. Clair Thomson's speculum, has been cut through by a sweep of Ballenger's swivel knife along the heavy-dotted line. [From the Author's *Diseases of the Nose.*]

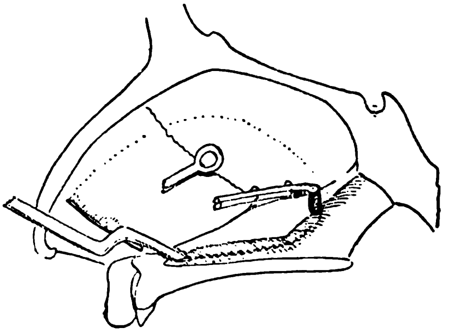

FIG. 4.—Diagram shewing line of fracture of the vomer. [From the Author's *Diseases of the Nose.*]

The second part of the operation concerns the vomerine deformity, and is the same whether spurs are absent or present. It may be pointed out here that if a patient is asked to undergo an operation which is not one of necessity but rather of expediency, he deserves to have a really adequate result; in a word, a surgeon must not remove an anterior and leave the patient with a posterior obstruction, because the latter was unexpected or difficult to deal with. If this course be adopted, the subsequent removal of the posterior obstruction will prove difficult. The deformed portion of the vomer is best removed by cutting through its anterior attachment to the intermaxillary with the gouge and mallet, while its upper edge, where it thins out behind a spur, must be severed with the punch forceps. Both these manipulations are easily carried out through the mucous membrane incision. It is troublesome and unnecessary to elevate the periosteum from the lower surface of a vomerine ridge. Adams's or Walsham's forceps are now introduced, one blade in each nostril, so as to grasp the vomer with its mucous membrane intact. With a few turns of the wrist the bone will, in all but young persons, be made to crack through near its attachment to the palate, and when the detached bone is now inspected through the incision it will be found lying almost free and requiring very little traction to bring it away without tearing the soft tissues. With the punch forceps all remnants of the vomer and of the perpendicular plate are snipped away until the middle line is reached at all points. That this end has been attained is only determined by a final examination of each nasal fossa. The large mucous membrane sac is carefully searched for loose fragments of bone, and the operation is completed by introducing a rubber splint on either side to prevent accumulation of blood between the two sheets. No stitching is required. In bad fracture cases it is difficult not to tear the tissues, but unless such tears occur on corresponding parts of the two sheets, no perforation will result. The patient should be put to bed for twenty-four hours, and should follow the injunctions as to peroxide, cold applications, and avoidance of alcohol as detailed on page 46. The splints will be removed in twenty-four hours; this is best done by catching each with Spencer Wells's forceps and twitching them out at the same moment by a sudden downward movement. Should they be expelled by sneezing, they must not be re-inserted except by the surgeon; I have known a nurse re-introduce a splint between the two flaps. The results of this operation appear to be perfectly good. Perforation occurs in exceptional cases from injuries done during operations, but never from sloughing of the flaps. I have seen one case of fairly free haemorrhage from the naso-palatine artery which was easily controlled by plugging, and one case in which uncomfortable

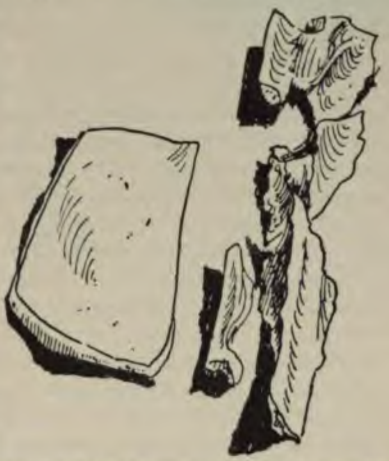

Fig. 5.—The main fragments from a submucous resection of the septum. The square portion of cartilage removed with the swivel knife, and portions of the distorted vomer (natural size). [From the Author's *Diseases of the Nose*.]

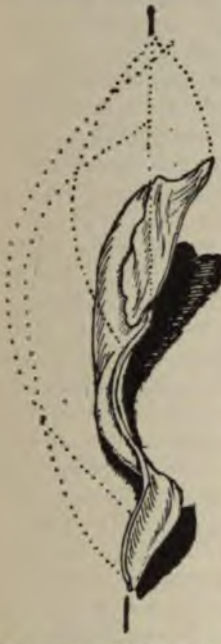

Fig. 6.—Sigmoid lower edge of septal cartilage: fragment from a submucous resection operation. The dotted lines indicate the planes of the septum which blocked the anterior two-thirds of the right and the posterior third of the left half of the nose. [From the Author's *Diseases of the Nose*.]

sensations resulted from the implication of the naso-palatine nerve in scar tissue. In developmental cases originating early in life one vestibule may be deficient in size, and failure to restore two perfectly free nostrils may result.

HAEMATOMA AND ABSCESS OF THE SEPTUM.—**Haematoma** of the septum is always traumatic, generally bilateral, and is seldom seen except in children. The history given is that of pronounced nasal obstruction, speedily following a blow on the nose. On examination one or both nostrils can be seen to be blocked by a smooth, rounded, red swelling immediately behind the vestibule of the nostril. It is soft and fluctuating, and its origin from the septum and not from the outer wall must be determined by the use of the probe. A much-enlarged anterior end of an inferior turbinal or the lower end of an old nasal polypus, on the surface of which a close network of capillaries has developed after long exposure in the air-way, may conceivably be mistaken for this condition. In a healthy child, if no abrasion of the mucous membrane occurs at the time of injury or through subsequent use of the finger-nail, the blood will be absorbed, and in the course of two or three weeks the septum will have returned to the normal. During the early days the temperature must be carefully watched, for in a good many cases suppuration occurs, and it may be said that abscess of the septum almost always has a traumatic origin.

It is difficult to diagnose the presence of pus by inspection of the tumour, and if any of the symptoms of suppurative inflammation are recognised, incision must be made without any delay; and it is wise to err on the side of over-activity, for if an abscess be allowed to remain unopened for a few days, necrosis of cartilage may result, and external deformity follows when cicatrisation takes place. When the swelling is bilateral a very large incision should be made on both sides under gas anaesthesia. The knife must go right down to the cartilage. The lips of the incision unite with remarkable rapidity, and it is essential to prevent this in order to secure perfectly free drainage. Drainage tubes and rubber tissue are speedily extruded from the wound, and the best method is to introduce a hank of horse-hair tied, as in the sketch, so as to be self-retaining. This drain must be kept in until healing by granulation is well advanced.

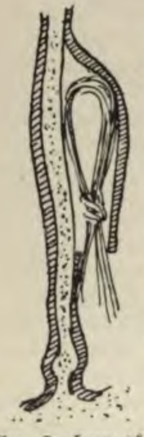

FIG. 7.—Loop of horse-hair maintaining drainage in abscess of the septum following haematoma.

In exceptional cases abscess of the septum has been recorded as following typhus, small-pox, dental disease, and so forth, and in others as occurring idiopathically.

PERFORATION OF THE SEPTUM.—With very rare exceptions, which are ascribed to severe inflammations accompanying zymotic diseases, perforations fall into one of four categories, namely, those due to syphilis, lupus, injury, or perforating ulcer. The first two are dealt with under special headings elsewhere (p. 27), but it may be stated here that syphilitic perforations are the result of tertiary or gummatous infiltrations, and that this is the only disease in which any destruction of the bony elements occurs. A perforation of the septum, in which a portion of the vomer or perpendicular plate is missing, is certainly due either to injury (generally operative) or to syphilis.

Perforation resulting from lupus is quite common, and occurs in the quadrilateral cartilage near the orifice of the nose. It is not improbable that the frequency with which lupus appears first in this locality is due to infection or damage of the part by the finger-nail. The disease first infiltrates and pierces the cartilage, making its appearance in the opposite nostril. When a perforation has subsequently formed, as it commonly does after scraping or burning operations, it is found to be surrounded with the characteristic crust-covered granulations or else with cicatrised edges. (*Vide* p. 27.)

Perforating Ulcer of the Septum.—Upon the cartilaginous portion of the septum, immediately above the intermaxillary bone, there is an area of mucous membrane in which the network of capillaries is especially close, whilst the vessels themselves are large and thin-walled; epistaxis commonly occurs here spontaneously or as the result of slight injury. Small blood-clots may therefore adhere to the surface, and on the other hand interstitial haemorrhages may cause changes in the tissue, which appears atrophied and yellow in the cadaver (xanthosis of Zuckerkandl). Furthermore, the area receives the full blast of the inspiratory current of air, and this impinging upon it cold, dry, and dust-laden, is apt to impair the delicate epithelial covering. In certain individuals the epithelium here loses its ciliated character, and a dry film of dust-laden mucus clings to it. The little finger is instinctively introduced to remove this; abrasions and haemorrhages are created, and the blood-clots again call for the introduction of the finger-nail, and in time a shallow ulcer is formed, leading to further crusting, irritation, and scarification. It is an instance of a somewhat defective piece of tissue exposed both to unfavourable atmospheric and to traumatic conditions, and the result is ulceration and finally perforation. The whole course of events is exceedingly slow, and the resulting hole is circular with thin, cicatrised edges. Occasionally these perforations give trouble by the collection of dried mucus upon the lower edge, but the patient is often unaware of the existence of any abnormality. During the ulcerative or pre-ulcerative stage, the best treatment is the application, upon a wool swab, of an ointment such as the ung. hydrarg. ox. flav. dil.; the use of the finger-nail must be rigorously avoided, thick gloves being worn at night for a time to prevent its unconscious practice.

New Growths.—All the usual forms of tumour may occur upon the septum, but they are very far from common; enchondroma, fibroma, angioma, lymphoma, papilloma, carcinoma, and sarcoma have all been recorded. The most characteristic septal tumour, however, is the so-called bleeding polypus. This may be described as a pedunculated, dark-red, mammillated growth attached to some part of the cartilaginous portion. It is highly vascular, and is the source of severe attacks of recurrent spontaneous epistaxis. If partially removed it speedily regains its original size, which may be larger than that of a filbert; on this account it was at one time classed among the sarcomas. If thorough removal is followed by cauterisation of the point of origin, no recurrence takes place, and secondary dissemination has never been recorded. Microscopically there is some variation in the relative proportion of the component tissues and in the degree of maturity of the connective tissue, but in general the designation fibro-angioma is applicable. Certain cases with both the appearance and the symptoms of bleeding polypus have turned out to be instances of lupus.

ERNEST WAGGETT.

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E. W.

RHINOSCLEROMA

By HERBERT TILLEY, B.S., F.R.C.S.

This rare disease was first described in 1870 by Hebra, who applied the name to a chronic progressive induration of the tissues in the immediate neighbourhood of the nostrils and upper lip.

Etiology.—Little is known of the conditions under which rhinoscleroma originates except that it occurs almost entirely amongst the poor and unclean of both sexes. The majority of cases have occurred in the south-east of Europe, especially Russian Poland, Galicia, and Hungary, and a few cases have been reported in Central America, Egypt, and India. Sporadic cases amongst the alien poor of London have also been reported (6). It is very doubtful whether the disease is contagious; inoculation experiments have given negative results.

Morbid Anatomy and Pathology.—The sites of predilection of the scleroma are the cartilaginous parts of the nose, the commencement of the bony part of the nasal cavity, the posterior choanae, and the larynx below the glottis. Each of these places may be affected independently, and not by extension from one part to the other. The disease may be regarded as a chronic infective granuloma, that is to say, a round-celled infiltration which rapidly develops into fibrous tissue possessed of great power of cicatrisation, so that considerable deformity of the affected parts frequently results. There are also numerous larger cells and spaces—"vacuoles"—formed by hyaline degeneration of larger cells. Frisch was the first to observe in the larger cells and "vacuoles" bacilli which stain by Gram's method, and in many respects resemble Friedländer's pneumo-bacillus.

The symptoms necessarily vary according to the part affected. Out of eighty-five cases the mucous membrane of the nose was attacked in eighty-one, the cutaneous covering of the nose in seventy-four, the pharynx in fifty-seven, the larynx in nineteen, the trachea in five, the upper lip in forty-six, the upper jaw in sixteen, the hard palate in seventeen, the tongue in four, the lower lip in two, the lacrymal tract in five, and the ear in one case. When the nose is affected, the chief symptom is chronic, progressive, and painless obstruction, which is brought about by the smooth, pale nodules which grow in the infiltrated and cicatrising mucous membrane. In the earlier stages of the intranasal disease there may be some muco-purulent discharge and crust-formation; bleeding is uncommon and ulceration rare. If the skin of the nose be affected, that feature becomes swollen, indurated, and disfigured (*vide* Figs. 22, 23, pp. 140, 141); brawny desquamation may occur from atrophy of the sebaceous and sweat glands. The nostrils are frequently reduced to mere slits by the growth within the vestibules of the hard, smooth nodules so characteristic of the disease. In course of time, and the process may be extremely slow, the disease spreads downwards from the naso-pharynx to the soft palate and pharynx, and as a result of infiltration these structures lose their normal functions, whilst cicatrisation may result in extreme narrowing of the passage between the throat and naso-pharynx. In the larynx the subglottic region is most frequently affected, and tracheotomy may be necessary to obviate the stenosis and consequent dyspnoea. (*Vide* Plate V.)

There is strong evidence that Stoerk's blenorrhoea is scleroma of the upper air-passages. (*Vide* also p. 140.)

Diagnosis.—Rhinoscleroma may be distinguished from lupus, tuberculosis, malignant disease, and syphilis by its slow progress, its characteristic induration and painlessness, and by the absence of ulceration and of offensive discharge. If the appearances suggest the possibility of syphilis, antisyphilitic treatment will soon render the diagnosis clear. In very doubtful cases the diagnosis of scleroma could be settled by detection of the characteristic bacilli in portions of the infiltrated area or by microscopic examination of the diseased tissues.

Prognosis.—The disease is very chronic, and may last for more than twenty years. The only dangerous variety is that which attacks the larynx. Complete involution of the growth has followed an attack of typhus fever, and in a second case an attack of malarial pyrexia.

Treatment.—The internal administration of drugs has no effect on the disease. Good results have been reported from injections of 1 to 12 per cent solutions of arsenic into the affected part, and a 2 per cent solution of carbolic acid has been used in the same way; such treatment may have to be continued for many months. Complete nasal obstruction is most satisfactorily relieved by free excision of the indurated tissues followed by the insertion of hollow bougies. Recurrence of the growth nearly always follows, and frequent intervention may be called for. Tracheotomy must be performed when dyspnoea is caused by laryngeal stenosis.

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GLANDERS

By HERBERT TILLEY, B.S., F.R.C.S.

This rare disease is caused by the direct inoculation of those whose occupation brings them into contact with horses suffering from glanders. The specific organism, the *Bacillus mallei*, occurs in large numbers in the nasal mucus of the infected animal. The organism may gain access to the human system through an abrasion of the skin or by way of the nasal mucous membrane; in a large proportion of cases the nasal mucosa is first affected.

Symptoms.—The nose and soft tissues in the immediate neighbourhood become swollen, red, and tender, and the adjacent lymphatics and the cervical glands greatly inflamed, enlarged, and frequently suppurate. The nasal discharge, which at first is clear, quickly becomes yellow, fetid, and blood-stained. Examination of the nasal cavities in the early stage reveals the presence of small, pale, nodular elevations; these soon become red, then yellow, and break down, so that ulcers are produced, which rapidly unite and destroy mucous membrane, cartilage, and bone. The accessory nasal sinuses may be similarly attacked. The *Bacillus mallei* may easily be detected in the purulent nasal discharge. The constitutional symptoms are very severe, and towards the end present a pyaemic character.

Prognosis.—Acute cases are almost invariably fatal within ten days. In rare instances the disease may become chronic, and about half the cases recover.

Diagnosis.—The intense aching of the limbs in the early stage may closely simulate rheumatism, but the appearance of the eruption would exclude that disease. The presence of the nasal discharge and acute symptoms in a patient whose work is amongst horses should always arouse the suspicion of glanders, and if there be any doubt as to the nature of the disease, the specific organism should be sought for in the nasal discharge, or inoculation experiments carried out upon guinea-pigs.

Treatment.—Beyond supporting the patient's strength and keeping the nasal cavities cleansed by warm alkaline antiseptic washes, little can be done to combat the disease. It is obviously important to warn stablemen, farmers, and other exposed persons of the dangers they run when brought into close contact with a "glandered" horse; they should be urged to protect any cuts or abrasions on their faces or hands carefully, and to be especially cautious not to soil their handkerchief or clothing with any of the nasal mucus from the infected animal. (For further information the reader is referred to the article "Glanders" in Vol. II. Part I. p. 201.)

HERBERT TILLEY.

FOREIGN BODIES IN THE NOSE

By HERBERT TILLEY, B.S., F.R.C.S.

Foreign bodies may find their way into the nasal passages in various circumstances. Children frequently put foreign bodies into their nostrils, and hysterical women and lunatics may do the same. Bullets and portions of knives and other sharp instruments penetrating the skin have thus entered the nasal fossae. Plugs introduced to control epistaxis have occasionally been forgotten. In some cases a foreign body has been forced into the naso-pharyngeal cavity in the act of vomiting. Among the most common articles met with in the nose are fruit-stones, buttons, beads, pieces of wood or slate pencil, shells, or pebbles. A supernumerary tooth sometimes erupts into the nasal cavity.

Symptoms depend in great measure upon the nature, size, and shape of the foreign body. As a rule, the presence of a foreign body in the nose sets up a discharge which at first is muco-purulent, but it may become fetid and tinged with blood. In most cases there is local pain, which may radiate over the side of the face. Where the foreign body is large, or has caused much swelling, there is obstruction of the affected nostril. There is often sympathetic disturbance of the eye and ear, as shewn by increased secretion of tears, earache, tinnitus, and even otitis media. The voice may lack its usual resonance, and attacks of sneezing, giddiness, and vomiting have been described. Delirium has occurred in a child. In process of time toleration may be established, and instances have been recorded of foreign bodies lying in the nose for many years without giving rise to any marked symptoms.

Diagnosis.—The existence of a unilateral purulent, fetid, or bloody discharge from the nose, especially in a child, should always lead to the suspicion of a foreign body. If there be any doubt in the matter, careful cleansing of the nose, and the use of the probe after cocainisation of the nasal mucous membrane, will usually clear up the diagnosis. In children it may be necessary to give a general anaesthetic in order to make a satisfactory examination without injury to the soft parts.

Treatment.—If the foreign body be small, non-absorbent, and recently introduced into the nasal cavity, an attempt may be made to expel it by one of the pneumatic methods. The simplest method is to make the patient sneeze by tickling the inside of the occluded nostril or by inhaling a small quantity of snuff; during the act of sneezing the free nostril should be stopped, so that the full blast of air passes down the obstructed nasal cavity. Again, the patient may be asked to close the lips tightly and distend the cheeks with air, while the surgeon drives a sudden blast of air round the nasal cavities by means of a Politzer's bag. The same method can be applied to a crying or screaming child, and in such a case the air should be driven into the free nostril during the expiratory phase of the crying. If such methods fail, a 10 to 15 per cent solution of cocaine should be sprayed into the obstructed nasal cavity, and when the parts are insensitive, an attempt should be made to extract the foreign body with a loop of wire, a curved probe, a strabismus hook, or suitable nasal forceps. In young children a general anaesthetic will almost always be required.

It is unwise to attempt to remove a foreign body from the nose by syringing lotions up the unobstructed side, for the danger of the fluid entering the tympanum, and causing inflammation therein, is a very real one. The risk is more imminent when suppuration has taken place around the foreign body. HERBERT TILLEY.

RHINOLITHS

By HERBERT TILLEY, B.S., F.R.C.S.

This name has been applied to the deposition of calcareous matter within the nose, forming a stone or nasal calculus.

Etiology.—Women are much more subject to this affection than men. No obvious explanation of this preference is forthcoming; it has been suggested that women blow their noses less than men, and that consequently there is a greater liability in them to retention of secretion. Rhinoliths gradually increase in frequency above the age of ten; but they are occasionally met with under this age. Concretions of a characteristic form are frequently found in the nostrils of cement-workers; chiefly in those who rake out cement-ovens, and consequently inhale hot cement dust.

In the great majority of cases the concretion takes place round some foreign body, which may have been introduced into the nostrils, or may have entered through the choanae in the act of vomiting or sneezing. The nucleus may consist of a bead, button, or other foreign body. In some cases it is represented by a piece of inspissated mucus or a blood-clot. Three conditions seem to promote the formation of these concretions: (1) an abnormal condition of the nasal and lacrymal secretion; (2) any condition, such as nasal stenosis, which leads to retention of the secretion; (3) the presence of micro-organisms; this last condition may depend upon the two former. Micro-organisms attack the lime salts of the nasal mucus and favour their deposition on the foreign body. Usually one stone only is found; and the exceptions to this rule are more apparent than real, the second stone being probably a small mass detached from the first in the process of extraction. Cases, however, have been recorded in which two stones have been found; and in one or more cases a stone has been found in each nostril. The usual weight of rhinoliths is from 7 to 90 grains; a stone weighing

720 grains has been recorded. In colour, rhinoliths vary from a dirty white to grey, brown, or black. They may be soft and crumbling, or as hard as ivory. Chemically, rhinoliths are composed chiefly of the phosphates and carbonates of calcium and magnesium, with traces of the chloride and carbonate of sodium, and a certain proportion of organic matter. Traces of iron have been detected occasionally, probably in cases in which the nucleus was composed of that metal.

The symptoms due to the presence of a rhinolith are similar to those caused by a foreign body in the nostril. Inasmuch, however, as the rhinolith grows slowly, the symptoms come on slowly; there is usually more or less complete nasal obstruction, associated with a unilateral, muco-purulent, fetid, and occasionally blood-stained discharge. In exceptional cases, in which the septum has become perforated, there may be discharge from both nostrils.

Diagnosis.—The nostrils should first be cleansed with a warm alkaline antiseptic, and the obstructed side anaesthetised with a 15 per cent solution of cocaine. Careful anterior rhinoscopic examination, assisted by a probe, will generally enable the presence of a rhinolith to be determined. The probe must be used gently, in order not to induce free bleeding from the granulations which usually surround the concretion. There should be little difficulty in excluding atrophic rhinitis, simple polypus, malignant disease, and syphilitic sequestra.

Prognosis.—The removal of the rhinolith is almost invariably followed by an immediate cessation of the symptoms it has produced.

Treatment.—If the rhinolith be small, an attempt should be made to remove it by means of forceps, or a suitably curved probe, and cocaine anaesthesia will probably suffice. (*Vide* Foreign Bodies in the Nose, p. 57.) If, on the other hand, the concretion be very large, an attempt may be made to crush or fracture it within the nasal cavity, and to remove it piecemeal. If these measures fail, a general anaesthetic should be administered, and the cartilaginous portion of the nasal septum divided along the greater part of its lower attachment, for by this means a surprising amount of extra room will be secured, and a large intranasal tumour may be removed. Beyond frequently spraying out the nasal cavities with a warm alkaline lotion during the few days which follow the operation, no after-treatment will be necessary.

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H. T.

MAGGOTS IN THE NOSE

By F. DE HAVILLAND HALL, M.D., F.R.C.P.

This disease is almost entirely confined to the tropics; a very small number of cases have occurred in Europe. In India, where the disease is called "Peenash," it is fairly common; but this name is used rather loosely to include several affections of the nose not necessarily indicative of the presence of maggots.

The fly is the *Lucilia hominivora*, or the *Chrysomyia macellaria* Fabricius; to the larva of the latter the name "screw-worm" has been applied (*vide* Vol. II. Part II. p. 185). In rare instances the larvae of other flies have been met with in the nose.

The fly commonly enters the nostril during sleep. As a rule, patients suffering from ozaena are attacked. The flies are probably attracted by the smell, and they find a ready entrance into the capacious nostrils of patients with atrophic rhinitis. It would appear that the larvae are deposited in healthy nostrils accidentally.

The larvae develop very quickly and in enormous numbers. In one case 388 maggots were counted. The symptoms which first appear are excessive irritability of the pituitary membrane, sneezing, and a sanious discharge from the nostrils. In some cases epistaxis occurs. In severe cases intense frontal headache, anorexia, and fever with delirium are met with. The nose and face are swollen, and the larvae may be seen escaping from the nostrils. Not only the mucous membrane of the nose, but even the cartilages and bones of the nose may be destroyed, so that death may occur from septic meningitis either of hæmic origin or due to direct extension from the sphenoidal and ethmoidal sinuses invaded by the larvae.

The **prognosis** is always a grave one; for instance, out of seven cases occurring at Fort Clarke, in Dakota, all except one were fatal. This is, however, an exceptionally high mortality.

Treatment.—The only effectual method of treatment is the use of chloroform. At the very beginning of the disease, and in slight cases, the inhalation of chloroform may suffice. In more severe cases the patient must be anaesthetised, and the nostrils syringed out with a mixture of equal parts of chloroform and water, or with pure chloroform. Spraying the nose with a one in forty solution of carbolic acid in oil will relieve the pain produced by the injection of pure chloroform.

Centipedes, caterpillars, earwigs, leeches, and ascarides have occasionally been known to take up their abode in the nose. The symptoms produced are those common to the presence of any foreign body, with the addition that the movements of the visitor give rise to excessive formication in the part. In the case of the leech, epistaxis has been noticed as a symptom.

F. DE H. HALL.

NASAL NEUROSES

By Sir FELIX SEMON, K.C.V.O., M.D., F.R.C.P., and P. WATSON WILLIAMS, M.D.

Olfactory Neuroses.—The olfactory nerve is the nerve of the special sense of smell. Numerous nervous filaments derived from the olfactory bulb pass through the foramina in the cribriform plate to the mucous membrane of the upper part of the septum and of the outer walls as far down as the middle turbinated body, and to the olfactory cells of Max Schultze, from which the fine terminal filaments pass through the external limiting membrane of v. Brunn, to lie between the columnar epithelial cells. The mucous membrane here is peculiarly soft, thick, delicate, pulpy, and highly vascular. For the normal perception of odours it is essential that the odoriferous particles should reach the mucous membrane of the upper part of the nasal passages, and that these should be in a moist condition ; thus, any local abnormality preventing inspiration through the nasal passages, or the presence of polypi or collections of mucus and secretions, or a permanently dry condition of the mucous membrane, will interfere with the sense of smell or completely destroy it.

The terminal filaments of the olfactory nerve may be impaired in various chronic conditions of the mucous membrane, inflammatory and degenerative ; thus, in chronic rhinitis, or as the result of irritating injections, sprays, or douches, there may be more or less defect of smell.

In testing the sense of smell it is imperative to exclude substances, such as ammonia, which act on the nerve of common sensation, lest we confuse olfactory and purely sensory impressions. Musk or some such scent should be used as the test.

Anosmia, or complete loss of the sense of smell, is frequently observed in polypus cases, as a consequence of disease of the fifth nerve ; or in the dry atrophic condition of the mucosa in atrophic rhinitis. It may be either unilateral or bilateral ; and may result from congenital defects, blows or falls on the head producing fracture of the cribriform plate, basic meningitis, intracranial tumours, syphilitic disease, embolism or haemorrhage of the middle cerebral artery, or from the less gross central lesions associated with epilepsy, locomotor ataxia, general paralysis, hysteria, and insanity. In these affections anosmia is frequently associated with loss of taste (ageusia). A few cases of unilateral destruction of the olfactory bulb with anosmia are recorded, and in these the left bulb has always been the one affected. Anosmia is very rarely detected in intracranial haemorrhages or tumours, as in the few cases in which the sense of olfaction is interfered with their effect is almost always unilateral. Anosmia has been observed to follow the removal of both ovaries.

Incomplete anosmia is commonly present when the mucous membrane of the upper portions of the nasal passages is swollen from irritation or from infection by pyogenetic organisms, or in nasal accessory sinus suppuration, or when a persistent nasal catarrh results from adenoid growths.

Parosmia, or perversion of the sense of smell, in which imaginary or subjective perceptions of odours are present, is usually of central origin. It occurs in hysteria, hypochondriasis, epilepsy, influenza, and lesions of the anterior temporal lobes ; it is important to realise that it may be the first sign, or one of the first signs, of mental derangement. Olfactory hallucinations have been known to occur in sexual neurasthenia.

Hyperosmia, hyperaesthesia of the olfactory nerve, with increased sensitiveness to smell, may arise in neurasthenic conditions with exaggeration of all nervous impressions, in hysteria and hypochondriasis, or as the result of irritative lesions affecting the olfactory bulbs.

The *prognosis* in anosmia will depend very much on the special cause of the loss of the sense of smell. If it be due to nasal polypi, or other removable causes, the prognosis is favourable, provided the loss of function have not persisted for a long time; after two years' continuance of complete anosmia the sense is seldom regained. But when associated with degeneration of the mucous membrane, as in syphilitic disease or atrophic rhinitis, the olfactory end-organs become atrophied sooner, and the prognosis is then hopeless. Loss of smell due to organic central nervous lesions will rarely be restored; on the other hand, the prognosis in functional anosmia and parosmia, except in cases in which this symptom precedes insanity, is generally favourable.

Treatment.—If the neurosis of olfaction be due to local disease this should be treated, and, with the removal of the cause, the functions of the olfactory nerve may possibly be restored; but very little more can be done. It is well to investigate thoroughly the possibility of any of the local conditions mentioned above causing anosmia, and to avoid making a hasty diagnosis of true neurotic anosmia. But in the latter affection local galvanism and faradisation may prove useful, and strychnine and arsenic may be given internally.

The treatment of anosmia and parosmia due to central nervous affections resolves itself into the treatment of the various causes of the disease. The purely functional cases should be treated by nervine tonics, change of air and rest; and in women any irregularity in menstruation should receive attention.

Sensory and Reflex Neuroses of the Nose.—A great deal has of late years been said and written in reference to the reflex neuroses of the nose. That these neuroses occur, and that their effect may be far-reaching, there is now no room for doubt. But while, on the one hand, there has been a tendency on the part of some clinicians to ignore the obvious existence of these diseases, there has been an unfortunate proclivity, on the other hand, to refer all and sundry obscure neuroses to the nose, and to explain their occurrence as nasal reflex phenomena. We may recall the views put forward by the late Professor Hack, who stated that the reflex irritability of the nasal mucous membrane was dependent upon the expansibility of "erectile organs" causing a sudden and transitory swelling of the anterior ends of the lower turbinals, and consequent excitation of the nerve-endings, and that then reflex irritation elsewhere occurred. Starting from the hypothesis that many nervous symptoms thus originated from the congested state of the cavernous tissue of the nose, he reasoned that by operative removal of the erectile tissue, the supposed connecting link, it might be possible to cure many of these obscure neuroses, namely, spasm of the glottis, nightmare, asthma, attacks of spasmodic cough, migraine, supraorbital and other forms of neuralgia, amblyopia and amaurosis, erythema or pseudo-erysipelas of the nose, giddiness, epileptiform attacks, secretory neuroses, ciliary scotoma, headache, hay-fever, and so forth. To this list his followers added Graves' disease, diabetes, irregularity of the heart's action, tachycardia, stenocardia, angina pectoris, cardialgia, diseases of the stomach, dysmenorrhoea, chorea, enuresis nocturna, melancholia, neurasthenia, catarrhal affections of the air-passages, epiphora, hyperaemia and oedema of the conjunctiva and the eyelids, blepharospasm, asthenopia, narrowing of the fields of vision, aural and genital disturbances, spasm of the facial and other motor nerves, muscular pains, stiffness of the neck, and cutaneous manifestations. The possibility that some of these neuroses are in a measure dependent on nasal abnormalities must be admitted, but such cases certainly are extremely rare. We would emphasise the importance of being on our guard against the error of exaggeration, while giving due regard to the large class of cases that may legitimately be included under the term *nasal neuroses.*

When we remember the intimate anatomical correlation between the nerves supplying the nose and other regions, near or more distantly situated, it is easy to conceive that centripetal impulses from the nerves in the nasal passages may have far-reaching reflex effects. The nasal mucous membrane is supplied with ordinary sensation by the ethmoidal branch of the nasal nerve and branches from Meckel's ganglion. This is connected with the Gasserian ganglion, which, in turn, is in relation with the carotid plexus of the sympathetic and perhaps with the pneumogastric. We would specially direct attention to Cajal's observations that a few of the collateral fibres from the gelatinous substance of Rolando, which is the receptive nucleus of the trigeminus in the medulla, break up under the motor nuclei of the facial and vagus, "and the inference is they communicate." Also in the mouse Cajal figures collaterals from the sensory ganglion in the substance of Rolando: the descending root of the fifth nerve terminates by arborisation in the substance of Rolando, and from the cells of the substance of Rolando fibres arise which terminate in the nucleus ambiguus of the same side and in that of the opposite side. These observations serve to explain the physiological association existing between the several portions of the respiratory tract, and the possible interdependence of nasal disease and bronchial asthma.

The arterial supply to the mucous membrane and to the erectile tissue of the turbinated bodies is controlled by vasomotor nerves from Meckel's ganglion, and is under the control of the vasomotor centres in the medulla.

The physiological nasal reflexes are sneezing, coughing, lacrymation, and vasomotor changes producing increased secretion. Their intimate relationship with other reflex areas is seen in the effect of bright sunlight on the eye producing lacrymation, coughing, and rhinorrhoea; and in the fact that particles of food in the larynx excite lacrymation as well as cough. In some susceptible persons, if certain portions of the nasal mucous membrane are irritated by a probe, sneezing and lacrymation ensue; and if the turbinals are irritated posteriorly, especially if the

Eustachian orifices are touched, cough is often excited. Dust and particles of foreign matter and irritating vapours produce similar effects. The specially sensitive spots—called hyperaesthetic areas—have been described as being situated on the posterior extremity of the inferior turbinal and the corresponding portion of the septum, at the anterior extremity of the superior turbinated body, at the anterior extremity of the middle turbinated and the corresponding portion of the septum; to these must be added the lips of the Eustachian tubes. Further, it has been shewn that irritation of the nose may produce arrest of respiration and syncope from temporary arrest of the heart's action; similar effects are sometimes observed as the result of strong odours. Unilateral exophthalmos and increased pulse frequency have been observed (Semon) to follow an operation for nasal polypi; it is questionable, however, whether these events stood in the relation of cause and effect.

Hyperaesthesia is generally associated with a more or less definitely abnormal condition of the nasal mucous membrane; often, however, no abnormality can be detected. It is usually associated with sneezing and rhinorrhoea, and is generally the immediate local factor in hay-fever, nasal cough, and so forth. It may be due to any irritation or catarrhal inflammation in neurotic subjects; or it may result from central or from reflex causes in the eye, the ear, or the digestive or genital organs.

Incomplete *anaesthesia* results from various chronic degenerative diseases of the mucous membrane, and in many cases of polypus. Total anaesthesia may be due to degeneration or destruction of the sensory nerves, as in cerebral tumour or in intracranial syphilis; or it may be functional, as in hysteria.

Nasal Cough.—Occasionally cases arise in which there is a hard, persistent, dry cough, which ceases during sleep, due to an exaggeration of the normal nasal reflex cough. It may sometimes be excited by touching the sensitive parts of the nasal mucous membrane with a probe. If there be no pulmonary disease, and no other cause for such a cough can be detected, the possibility of its nasal origin should be borne in mind. Post-nasal growths will also excite it sometimes; but in cases of this class it may be due to irritation in the ear.

Vasomotor Neuroses.—Vascular engorgement of the nasal mucosa and distension of the erectile tissues, especially of the turbinated bodies, occur in two forms: (*a*) periodic vascular swelling; (*b*) vascular engorgement with coryza (vasomotor coryza), and various reflex neuroses, as in paroxysmal sneezing.

Periodic vascular engorgement and swelling of the erectile tissues is generally associated with nervous prostration or imperfect digestion. The fulness of the nasal mucosa produces more or less obstruction in one or both nasal passages; it comes and goes, and is usually worse at night on going to bed. Very often an examination is made just at the time when the swelling has subsided and nothing abnormal can be detected. If the patient be seen while the nose is obstructed by the turgidity of the tissues the real nature of the affection is readily distinguished from hypertrophic rhinitis on applying a cocaine spray, when vasomotor swelling entirely disappears. In some cases, particularly in gouty and dyspeptic patients, the engorgement of the mucous membrane is accompanied by redness and swelling of the nose externally and flushing of the face.

These changes are prone to occur—(i.) in persons of the neurotic temperament who are run down from hard brain work; (ii.) in gouty patients; (iii.) from abuse of alcohol; (iv.) in neurotic women, especially at the menstrual period.

The treatment must be mainly directed towards improvement of the general health by nerve tonics, massage, cold baths, and out-of-door exercises. It is sometimes necessary to use the galvano-cautery, linear cauterisations being made over the turbinated bodies; and if chronic rhinitis exist, suitable local treatment will be required.

Paroxysmal sneezing may be due (*a*) to reflex peripheral irritation; (*b*) to a central neurosis. The physiological mechanism of sneezing may be briefly described as a reflex act brought about by irritation of the trigeminal nerve, either in the nasal passages or in other regions which it supplies. We have met with cases, mostly in neurotic women, in which it occurred in paroxysms of thirty or forty sneezes, especially on rising in the morning, and when the face was plunged into cold water; and similar cases have been frequently recorded. In a few cases there is no rhinorrhoea; but as a rule there is lacrymation and stuffiness of the nose followed by watery discharge, and a pain in the bridge of the nose. Prolonged attacks are very exhausting. Paroxysmal sneezing is generally due to a hyperaesthetic condition of the nasal mucous membrane, and is often set up by irritation from particles of dust.

Hay-fever is a form of paroxysmal sneezing usually brought about by the irritation of certain kinds of pollen. Some patients are peculiarly susceptible to the effluvia of certain animals, for instance, the cat, horse, or dog, and invariably suffer from sneezing or asthma when in proximity to these animals; others are similarly affected by peaches, violets, roses, musk, peppermint, ipecacuanha, lycopodium, and so on. The exciting causes of paroxysmal sneezing are such that although every one is continually exposed to their influence, yet comparatively few persons suffer; thus it is obvious that individual predisposition is necessary for the occurrence of the affection. Patients are almost invariably of the neurotic temperament, especially dwellers in cities; and there can be no doubt that, to some extent, the predisposition is hereditary and connected with the gouty diathesis. The causes being so general and yet paroxysmal sneezing being relatively so rare, a third factor must be necessary for its occurrence; and in many cases some local abnormalities will be found in the nasal passages, the most frequent being (*a*) hypertrophic rhinitis; (*b*) spurs and bony projections of the turbinals or the septum; (*c*) deviations of the septum; (*d*) polypi; (*e*) old post-nasal vegetations; (*f*) areas of hyperaesthesia in the nasal mucous membrane. It would, however, be a grave mistake to hold every small deviation from the normal condition, which may be found in the nose, as responsible for the occurrence of paroxysmal sneezing, and to expect a cure from its removal.

Thus it appears that paroxysmal sneezing is the conjoint result of three factors:—(i.) The predisposing neurasthenic constitutional state; (ii.) an external irritant; (iii.) sometimes a pathological state of the nasal passages.

Nasal Asthma.—By the law of irradiation of reflex action—by the extension, that is, of reflex action from nerves in which it first appears to neighbouring ones, by means of the communications between the different systems or groups of ganglionic cells—persistent irritation in the nose may result in spasmodic asthma. It has long been known that asthma is sometimes associated with intranasal disease, but it is only since Voltolini's classical case of asthma, which he cured by the removal of nasal polypi, that attention has been directed to the causal connexion between nasal disease and asthma.

Further, we have to consider that when nasal coryza results from the action of some irritant conveyed by the inspired air, it is probable that the lower air-passages, though in a less degree, are simultaneously exposed to its influence; thus the asthma may be due to the latter influence. When the nose is partially or completely obstructed, and respiration oral, the defective filtration and warming of the inspired air will injure the lower respiratory tract. In these cases restoration of the nasal functions by appropriate local treatment will save the bronchial mucous membrane from much of the irritation to which it was previously subjected.

Treatment.—In paroxysmal sneezing and asthma, as in all sensory and reflex neuroses of the nose, treatment should be mainly directed to overcoming the underlying neurasthenia by appropriate nervine tonics—such as phosphorus, iron, arsenic, valerianate of zinc—and by general hygienic measures. A combination of iron and arsenic in particular, in steadily increasing doses, often has very gratifying results.

There is no doubt that paroxysmal bronchial asthma may be set up by nasal affections, and may be cured by the removal of the nasal disease; but such cases are rarely encountered. In the course of a special discussion on this question at the Laryngological Society of London in 1899, only a very few cases of actual cure of considerable and lasting improvement were recorded. One of us (W. W.) has recently met with such a fortunate result after a double radical antral operation and extirpation of nasal polypi and diseased ethmoidal cells. Severe attacks of asthma had appeared in the course of a few years, the patient never having had any asthma or paroxysmal sneezing before, and with the increasing severity and frequency of the asthmatic paroxysms her life was almost unbearable. The patient has been completely free from asthma for over a year, and now leads a very active life.

The continued resort to the routine method of galvano-cauterisation of the supposed irritable areas in the nose, not for patients in whom any abnormal intranasal condition can be discovered, but for every one afflicted with spasmodic asthma, calls for some allusion to its doubtful utility in any case. We do not know of any constant irritable area, although at the Pan-American Congress, in 1892, Forstensson (according to Jurasz) stated that he had discovered the specific point of irritability in the upper part of the septum in no fewer than 400 cases of asthma. Temporary cessation or diminution of attacks of bronchial spasm very often follows after any operative measure in the nasal passages, even a simple cauterisation. Many years ago the observation of this recognised fact led one of us (W. W.) to hope that repeated and limited cauterisation of the nasal mucosa, especially of the septal tubercle and inferior turbinals, particularly where these areas were hypertrophic, would result in a permanent cure of the asthma, but the improvement was only temporary, and certainly not such as to justify the routine cauterisation of the nasal passages of asthmatic patients. The method has of late years, however, been advocated, especially by Dr. Francis, who reports many cases of lasting cure from such cauterisation. His thesis is "that all asthma depends upon a condition corresponding to inhibition of exchange which is dependent upon the respiratory centre, and that the stability of the centre can be affected by nasal treatment." Hence in all cases while removing any existing causes of nasal obstructions, he advocates cauterisation of the nasal mucosa, even when no intranasal abnormality exists. We are bound to confess that we are still very sceptical of the lasting beneficial results of simple cauterisation of the normal nasal passages of asthmatic individuals. But when there is oedema or undue fulness of the middle turbinals impinging on the septal cartilage, cauterisation of the redundant tissue will often relieve or inhibit paroxysmal sneezing.

The nasal conditions should, of course, be carefully investigated; and if positively morbid changes be detected, such as are reasonably likely to cause the reflex neurosis, these must receive appropriate treatment; but the discovery of a small spur on the septum or of a small amount of erectile swelling on the middle or lower turbinated bones should not be proclaimed at once as the undoubted cause of the malady. Caution is the more necessary in these cases as it is very difficult, and often impossible, to distinguish between the cases in which intranasal treatment is likely to prove beneficial and those in which but a temporary effect will be obtained, or none. On the other hand, we may say that a good many cases of paroxysmal sneezing will probably be improved by local treatment. If chronic hypertrophic rhinitis, or polypi, or any other manifest nasal disease be found, it should certainly be treated in the hope that the neuroses may be relieved thereby; but assurance of cure by nasal treatment cannot be given to patients even if the concomitant nasal affection be well marked, for we cannot remove a hereditary or an acquired instability of the nerve centres. Nevertheless, by nasal treatment we sometimes obtain most brilliant results and perfect relief from all symptoms, particularly in cases in which nasal polypi and bronchial asthma coexist.

In paroxysmal sneezing, when the only abnormality is erectile swelling and vascular injection of the mucous membrane, we may cauterise the swollen parts superficially. The best plan is to ascertain, before applying cocaine, if there are any sensitive spots, and then to cauterise these after the cocaine has been applied.

A method followed by one of us (W. W.), who first employed it on himself with most gratifying results, is to spray the nasal passages cautiously with an aqueous solution of biniodide of mercury of the strength of 1 part to 20. A cocaine spray should be used beforehand, but, as the cocaine is destroyed by the mercurial salt, it is necessary to relieve the pain which very rapidly ensues by a hypodermic injection of morphine. The solution is intensely irritating, and care is necessary lest it get into the eyes or into the throat. The mucous membrane of the nose becomes much congested and swollen. In about three hours the pain and swelling subside, and are followed by a simple nasal catarrh lasting two or three days. In suitable cases, if this be efficiently done at the onset of the symptoms of that form of paroxysmal sneezing distinguished by the name of hay-fever, the patient will remain free from symptoms throughout the season; and there are very few persons who have suffered from the affection who will not readily undergo this or any treatment that offers a fair prospect of relief. This method has the advantage of leaving the sense of smell unimpaired, and involves no destruction of tissue. It may have to be repeated the following year, but in some cases the relief has extended over several years.

Pollantin, a serum-antitoxin obtained by injecting the toxic substance isolated from the pollen of certain grasses into animals, was introduced by Dunbar in 1903, and has given favourable results in a number of cases. One of us (F. S.) in the same year made a series of experiments which led to the conclusion that the toxin is undoubtedly specific, that it produces hay-fever only in predisposed persons, and that the antitoxin produces immediate disappearance of the subjective, and after a few minutes great amelioration of the objective symptoms. Many others have reported their experiences, and there is no reason to doubt that while the serum is fresh and is applied frequently to the nasal mucous membrane or the conjunctiva, marked amelioration follows in a number of sufferers. Unfortunately, however, practical experience has shewn that this happy result is by no means universal, and it would even seem that some patients who have been greatly relieved during one hay-fever season by the persistent use of the remedy, receive no benefit during the following. A few have even stated to one of us (F. S.) that their symptoms were distinctly aggravated by the use of pollantin.

Cocaine should never be recommended as a routine method for the relief of paroxysmal sneezing. It tends to aggravate the condition after its transient effects have passed off. Moreover, serious symptoms of cocaine poisoning may suddenly declare themselves, even after the patient has to all appearances become quite accustomed to the use of the drug. (*Vide* art. "Cocaine," Vol. II. Part I. p. 968.) The same applies to adrenalin.

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Idiopathic rhinorrhoea is a name applied to an affection in which the prominent symptom consists in a profuse watery discharge from the nasal mucous membrane. Whilst the group of cases comprised under this heading are probably due to a number of etiological factors, they are all essentially vasomotor neuroses. In some cases the copious discharge is the only symptom; in others it is accompanied by sneezing and lacrymation, and is in fact a form of paroxysmal coryza and sneezing in which the coryza is the prominent feature. In most cases the patients are of neurotic temperament; physical shock, hard brain work, exposure to cold, are the chief exciting causes to which it has been attributed; but in other cases it comes on suddenly without apparent cause. In a case recorded by Althaus it was associated with anaesthesia of the regions supplied by the fifth cranial nerve. In a case recently reported it was associated with disease of the pituitary body; usually, however, its cause has remained in obscurity.

The symptoms may begin with some itching or pricking sensations in the nose. When the discharge has continued for some hours the mucous membrane becomes swollen and oedematous. The copious, clear, colourless or slightly yellow discharge consists of water with traces of chloride of sodium and mucus. The amount varies very much; in some cases as much as two or three quarts have come away in twenty-four hours. In the intervals between the periodic attacks the mucous membrane resumes its normal aspect; but when the affection has existed for a considerable time, the mucosa becomes sodden and there is a tendency to the formation of mucous polypi. The disease may recur for months or years, but eventually it nearly always ceases spontaneously.

Cerebrospinal Rhinorrhoea.—The spontaneous escape of cerebrospinal fluid from the nose was first fully described by Dr. St. Clair Thomson in 1899. He shewed that cerebrospinal fluid can escape from the nose, and that this flow may go on uninterruptedly for years without visible impairment of health; and several cases collected by this author seem to indicate that this affection had been described hitherto in many cases as simple watery discharge from the nose. The discharge of fluid is generally from one nostril, and it persists night and day without cessation for weeks, months, or years. When intermissions do occur, the cerebral symptoms, headache and so forth, which often precede or accompany this affection, recur or are aggravated. There is no accompanying lacrymation or photophobia. Dr. Thomson's case was that of a female, aged twenty-five, in whom the escape of fluid from the left nostril persisted for over three years. There were short intermissions, and at these times very severe headaches occurred over the left eye and back of the head. In 1901, in conjunction with Mr. Stocker, one of us (W. W.) shewed a case at the annual meeting of the British Medical Association.

The patient has now remained quite cured and in perfect health for over two years.

Diagnosis.—The differential diagnosis between cerebrospinal rhinorrhoea and nasal hydrorrhoea depends mainly on the chemical reactions and composition of the escaping fluid, though the character of the symptoms also gives some indications of the nature of the disease. Prof. Halliburton's analyses (cited by Thomson) may be contrasted as follows:—

Cerebrospinal fluid.	*Nasal hydrorrhoea fluid.*
Is perfectly transparent, like water, contains no sediment, is not viscous.	Is thick and viscid, and slightly opalescent.
It is faintly alkaline, and is either tasteless or slightly salt. It gives no precipitate (mucin) with acetic acid.	Histological examination shews the presence of amorphous matter and mucous corpuscles. It gives, with acetic acid, a stringy precipitate (mucin).
Sp.gr. 1005 to 1010.	
Cold nitric acid gives a precipitate which disappears on heating.	
Saturation with magnesium sulphate or sodium chloride gives a precipitate.	
On boiling, there is not more than a trace of coagulum of serum globulin and serum albumin.	On boiling this precipitate with dilute sulphuric acid, a reducing sugar-like material is formed. This is also characteristic of mucin.
When boiled with Fehling's solution there is a reduction of copper (due to pyrocatechin or some similar body).	The fluid contains a small amount of protein coagulable by heat; *it does not reduce Fehling's solution.*
	Proteoses and peptone are absent.
The reducing substance may be obtained in needle-like crystals by evaporating to dryness an alcoholic extract of the fluid.	The presence of mucin and absence of reducing substance, as well as the considerable percentage of proteins and solids, are quite sufficient to distinguish this fluid from normal cerebrospinal fluid.

The symptoms may also be contrasted:—

Cerebrospinal fluid flow.	*Nasal hydrorrhoea.*
Headaches precede or follow the flow, but cease or are diminished during the persistence of flow.	Feeling of malaise sets in with the discharge, and disappears with its cessation.
No nasal symptoms.	Often ushered in with sneezing, lacrymation, etc.
It persists night and day.	It rarely continues during sleep.
The fluid drying, leaves the handkerchief pliable.	The fluid drying, leaves the handkerchief stiff.
No treatment is admissible.	Disease is amenable to treatment.

No *treatment* of any kind is, as a rule, of any avail. The case recorded by one of us (W. W.), however, became cured, and the cure followed some time after repeated lumbar puncture and withdrawal of cerebrospinal fluid. This procedure was suggested by Prof. Osler, but it is questionable whether it really influenced the cure, as the dropping of the fluid was not obviously diminished during the treatment.

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Coryza oedematosa is very closely allied to idiopathic rhinorrhoea. It consists of a serous infiltration into the connective tissue of the inferior and middle turbinated bodies, which is sometimes migratory and suddenly appears in other regions supplied by the trigeminus as it leaves the nasal passages. It is apparently connected with some irregularity of digestion in neurotic subjects.

No treatment appears to have any lasting results. Galvano-cauterisation of the turbinals may give relief, but local treatment is generally without any lasting effect. General hygienic measures and constitutional treatment should be adopted.

FELIX SEMON.
P. WATSON WILLIAMS.

3. DISEASES OF THE ACCESSORY SINUSES OF THE NOSE

By HERBERT TILLEY, B.S., F.R.C.S.

Introduction.—The study of the diseases of the nasal accessory sinuses is of comparatively recent origin, and possesses an importance not yet sufficiently recognised by our profession. By far the commonest morbid condition affecting these cavities is chronic suppuration, to the consideration of which this article will be almost entirely confined.

A suppurating focus may be regarded from at least three points of view: (1) the amount of pus secreted; (2) the effect exerted by the focus on the tissues in its immediate vicinity; (3) the general constitutional effect produced by the continuous absorption of septic discharges.

(1) In the case of the nasal accessory sinuses it is extremely difficult to state how much pus is poured out daily by any one sinus, but it is quite common to find a large cavity, for example, the antrum, overflowing with pus within three or four hours after it has been thoroughly emptied and cleansed. Since suppuration often occurs in several sinuses at the same time, it is obvious that at least two or three ounces of pus must be swallowed in the course of the twenty-four hours.

(2) The effect of a purulent focus upon the tissues in its immediate

neighbourhood will vary with its situation; for example, an antral abscess may induce few gross local changes, whilst suppuration within a sphenoidal sinus may cause blindness or other ocular symptoms, and is probably the unsuspected cause of many cases of septic basal meningitis.

(3) The evil effects of chronic nasal suppuration upon more remote regions, and on the general health, are now recognised. Dr. William Hunter has proved that certain pathological conditions may be produced in the gastric and intestinal mucous membranes by the constant swallowing of pus, and has also shewn that some of the graver forms of anaemia may be traced to the same source. Surgeons who devote their time to diseases of the upper respiratory tract are well aware of the subtle effect which nasal suppuration exerts on the nervous system; mental depression, apathy, inability to concentrate the attention, subjective feelings of weight and pressure in the head are common, and it is very remarkable how quickly these symptoms disappear, and how the whole demeanour of the patient changes when the purulent foci have been cured or efficiently drained.

Unfortunately the acquisition of practical knowledge on this subject is beset with difficulties; for although the nasal cavities are comparatively simple in their anatomical arrangement, the accessory sinuses which communicate with them are always complicated, and variations from the normal seem to be infinite. These dark recesses can be illuminated only by a skilfully directed pencil of light, and their examination demands from the surgeon a close apprenticeship, special technique, minute attention to detail, and not infrequently a rare degree of patience.

As already stated, this article will be confined almost entirely to the discussion of suppurative lesions. For the rest, it will suffice to bear in mind that fibrous, bony, cystic, and malignant growths may occur.

The accessory sinuses of the nose comprise the maxillary antra, the ethmoid, frontal, and sphenoidal sinuses. Each of these air-spaces communicates with its corresponding nasal cavity by at least one opening, and they are lined by ciliated mucous membrane, which is directly continuous with that of the nasal cavity. The close continuity of mucous surfaces involves the participation of the nasal sinuses in those inflammatory conditions which originate in the nasal mucosa. In view of the exposed position of the nasal mucous membrane, the varying temperature and the numerous impurities of the inhaled currents of air, it is not a little surprising that inflammation is comparatively infrequent. This immunity is probably due to many causes, such as the action of the cilia, which gives no rest to inhaled particles, but sweeps them downwards and backwards in the free secretion of mucus, which has been shewn by Drs. St. Clair Thomson and Hewlett, Park and Wright, and others, to exert an inhibitory influence on the growth of micro-organisms. Then, again, the highly developed and sensitive vasomotor mechanism, which controls the degree of distension of the vascular mucous membrane, subserves important functions in warming, moistening, and filtering the inspired air.

INFLAMMATION OF THE NASAL ACCESSORY SINUSES

Etiology.—*Acute Infectious Disorders.*—It is now fully recognised that acute and chronic suppurations are frequently due to infection by the specific micro-organisms of the acute infectious diseases. Not only has influenza proved a prolific source of sinus inflammation, but scarlet fever, enteric, pneumonia, diphtheria, whooping-cough, measles, and erysipelas may be the cause of the sinus inflammation. In some instances pure cultures of the specific organism have been obtained from the inflamed sinus, although as a general rule a mixed infection is found. Sometimes a patient dates the trouble from a very severe "cold in the head." In these circumstances, it is probable that the acute inflammation of the sinus produced such swelling of the lining mucosa that free drainage of the inflammatory contents became impossible, and the access of pyogenetic or specific micro-organisms set up suppuration. Such a course of events would be encouraged by the presence in the nose of any pathological condition hindering free drainage; for example, certain forms of hypertrophic rhinitis, deviations, spurs, or crests of the septum, nasal polypi, enlarged middle turbinals, and so forth.

Trauma not infrequently forms the starting-point of sinus suppuration, and in this way the frontal sinus has been affected by the kick of a horse, the antrum by the unskilful use of the galvano-cautery in the nose, or by the extraction of a tooth. Intranasal operations have not been altogether blameless in this respect.

Dental disease frequently causes antral suppuration, and this is commonly brought about by the rupture of an apical abscess into the sinus. The second bicuspid and the first and second molar teeth are most apt to cause trouble, because their roots come into closer relation with the floor of the antrum than those of the other teeth.

Infection of one Sinus from Another.—Not infrequently the ethmoidal cells and the maxillary antrum are infected by discharge from the neighbouring frontal sinus, but we must be careful in such cases to distinguish between an antrum which acts as a reservoir of pus and one which may be generating the discharge.

Chronic suppuration is generally due to causes which hinder resolution of the acute inflammation, and in most instances deficient drainage is the chief factor. In this connexion it must be remarked that the natural openings of the nasal accessory sinuses are small, and, with the exception of the frontal sinus, badly situated for purposes of drainage. Further hindrances to efficient drainage may be caused by obstructive conditions within the nasal cavities. In other instances the virulence of the primary infection may so damage the lining membrane of the sinuses that complete recovery from the inflammation is impossible, and this result may be aggravated if the general health of the patient be impaired or his surroundings be unhealthy. In the case of the maxillary antrum, chronic suppuration may be maintained by the persistence of a dental abscess which was the cause of the primary inflammation. Opinions differ as to whether suppuration may be chronic from the beginning. Many patients are quite unable to remember when the discharge began, and it is quite conceivable that in a person subject to frequent "colds in the head," the nasal mucous membranes may become so hypertrophied and catarrhal that a mild infection would graft itself upon them, and a purulent discharge would take the place of the mucoid secretion without the intervention of any acute symptoms.

Bacteriology.—As already stated, the specific micro-organisms of the primary disease have been found in pure culture in the pus in some cases of acute sinus suppuration. Usually, however, there is a mixed infection, in which staphylococci, streptococci, and pneumococci predominate. With reference to this subject, Drs. Lewis and Turner have, amongst other conclusions, shewn "that the pus obtained from some cases of antral suppuration may contain organisms similar to those occurring in the buccal cavity; that occasionally bacilli distinctive of dental caries may be isolated from the pus of an antral abscess; that in the cases of chronic suppuration streptococci were found in 80 per cent, whilst in the more recent cases they occurred in 60 per cent; that in recent cases virulent organisms are met with twice as often as in cases of chronic suppuration; that clinical evidence supports the view that the antrum is more frequently infected by way of the nasal cavity, and that this opinion is corroborated by bacteriological investigation."

SUPPURATION IN THE MAXILLARY ANTRUM: ANTRAL ABSCESS, ANTRAL EMPYEMA.—Suppurative inflammation may be acute or chronic.

I. Acute Inflammation.—*Etiology.*—As already mentioned, infection may enter by way of the nose or result from pyogenetic inflammation around the root of a tooth. The history of the case and the accompanying symptoms will generally enable the mode of infection to be determined, and it is of some importance to ascertain this, because the prognosis is better in cases of dental than in those of nasal origin.

Morbid Anatomy and Pathology.—There will be congestion, swelling, and oedema of the lining mucosa, and this may be so great that the ordinary cavity of the antrum is practically obliterated. As a rule, similar changes occur in the mucosa of the corresponding nasal cavity. The exudation, at first serous, rapidly becomes purulent, and may be mixed with blood effused from the congested vessels.

Symptoms.—There is usually painful tension within the cheek, much aggravated by stooping, coughing, or straining, and acute pain may be elicited by pressure over the malar bone, canine fossa, or the molar teeth. Severe neuralgic headache is common, and frequently the pain is most intense in the corresponding supraorbital region. The temperature may rise to 103° F., and the patient usually feels ill and depressed. As a rule, the exudation soon finds its way into the nose and the local symptoms subside, but if retention of the inflammatory products should occur, all the local symptoms increase in severity; the cheek may become red, swollen, and tender, and the inner antral wall may bulge into the nasal cavity.

Diagnosis.—In addition to the symptoms enumerated, examination of the nasal cavity with reflected light will reveal intense congestion and swelling of the mucosa as compared with the healthy side, and pus may frequently be seen in the middle meatus.

Prognosis.—With efficient treatment, cases of dental origin generally end in recovery, but when infection has occurred through the nose during the course of influenza or one of the acute specific infectious diseases, the prognosis is not so good; in these cases the poison is probably more virulent and its general debilitating effect greater. Whatever the origin of the suppuration, the prognosis will be the better the sooner free drainage is established.

Treatment.—(*a*) In cases of dental origin the offending tooth must be removed, and the antrum freely opened through the diseased socket and its cavity irrigated thrice daily with warm boracic, carbolic (1 in 60), or other mild antiseptic lotion. As the discharge diminishes the irrigations may be practised less frequently, and in the course of ten days to a fortnight they may be dispensed with altogether, provided the discharge of pus has ceased. Hot fomentations applied to the cheek give great relief to the pain, although some patients experience more comfort from iced or cold applications. Aspirin in 10-grain doses every three or four hours will often allay the headache and neuralgic symptoms.

(*b*) In cases of intranasal origin rest in bed, aperients, and remedies suitable for the primary constitutional disease take the first place. The application of heat or cold to the cheek may give much relief. To encourage free discharge from the antrum a small piece of wool moistened with equal parts of a 10 per cent solution of cocaine and adrenalin chloride may be applied to the middle meatal regions in order to diminish the swelling and congestion of the mucosa. With the same object in view Dr. Lack advises scarification of the middle turbinal.

If symptoms of retention occur, the antrum should be perforated below the attachment of the inferior turbinal (*vide* Fig. 8), and the inflammatory contents washed out with warm boracic lotion; great relief may follow this small operation, which it may be necessary to repeat three or four times before the discharge of pus ceases.

There can be no doubt that many cases of acute antral suppuration of nasal as well as of dental origin recover spontaneously; but it is equally true that early and efficient treatment will materially hasten the cure and relieve many of the most painful symptoms.

Chronic Empyema of the Antrum.—The etiology has already been dealt with on p. 74. The condition is often bilateral, especially when infection has entered by way of the nose.

Morbid Anatomy and Pathology.—As a general rule the mucous membrane is thickened and infiltrated with small round cells, and there is hyperplasia of the subepithelial connective tissue. These changes may be general, but more commonly are particularly pronounced in certain regions; for example, in the alveolar region, the concavity of the malar bone, and in the neighbourhood of the ostium. In very chronic cases there are large papilliform projections of the mucosa—the so-called "polypoid degeneration." Patches of granulation-tissue may be found on the floor of the cavity, and sometimes polypi are associated with periostitic thickening of the underlying bone. Apart from syphilis, caries, or necrosis of the bony walls of the antrum, is rare, even though suppuration may have lasted for several years. Persistent discharge of pus into the nasal cavities may lead to the formation within them of hypertrophy of the mucous membrane, periostitic inflammation of the inner antral wall, and to nasal polypi if the ethmoidal regions be affected. Only rarely does the pus find its way to regions other than the corresponding nasal cavity, but I have seen an external suppurating fistula below the malar prominence.

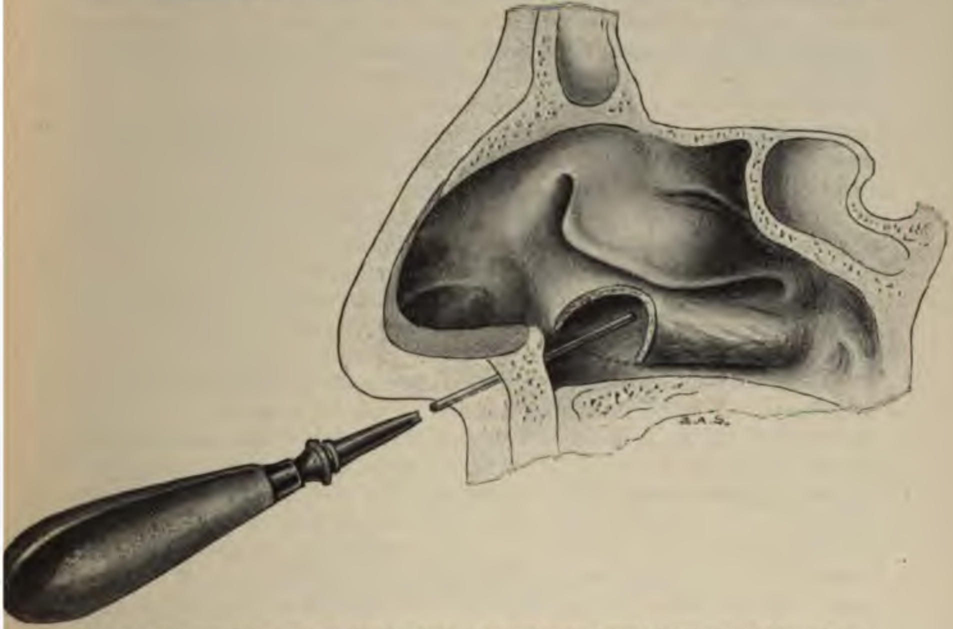

FIG. 8.—To illustrate exploration of the antrum. A small portion of the anterior end of the inferior turbinal has been removed to shew position of trocar when it has entered the antrum.

Symptoms.—The cardinal symptoms of a chronic antral suppuration are: (1) a purulent and frequently offensive discharge from the nose; (2) supraorbital headache; (3) more or less nasal obstruction.

The discharge is often first detected when the patient stoops; at night the pus is apt to pass backwards into the naso-pharynx, so that sniffing, screeding, and coughing are produced when the patient awakes, and frequently he thinks he is suffering from post-nasal catarrh—a diagnosis with which the medical attendant too often agrees. The patient is conscious of the fetor so long as his sense of smell remains, but as a result of catarrhal changes in the olfactory mucosa this may be destroyed after some months of antral suppuration. Headache is most frequently supraorbital, and is usually worse during the early part of the day. This periodicity is often regarded as due to malaria when it occurs in people who have resided in climates where this disease is endemic. Less commonly the pain is felt at the side of the nose or in the malar region. Nasal obstruction is caused by congestion of the nasal mucosa induced by the constant irritation of the purulent discharge. The middle turbinal is often swollen, and upon its outer side the uncinate process may present a cushion-like swelling, which is extremely characteristic of chronic antral suppuration. When definite polypi are present, the ethmoidal cells are generally infected.

In addition to these symptoms, the following are frequently met with: cough and liability to colds in the head, anaemia, indigestion, general debility, anorexia, flatulence, and other gastric symptoms due to the continual swallowing of the purulent discharge. Mental depression, feelings of weight and pressure in the head, are manifestations of the evil influence exerted on the nervous system by absorption of the purulent discharge. Aural symptoms are infrequent, but I have met with two cases of chronic suppurative otorrhoea, in which the discharge only ceased when the corresponding antral empyema was cured.

Diagnosis.—On ascertaining the existence of the symptoms already mentioned the teeth must be examined; special care must be taken not to overlook the presence of a "dead" tooth, which may be recognised by the absence of the sensation of heat in such a tooth when a hot probe is held against its "neck"—the junction of enamel and dentine. Innocent as a gold-crowned tooth may look, it may be the sole cause of antral suppuration. The nasal cavity should be examined with special reference to the presence of pus in the middle meatus. If none be seen in this situation, the patient should be directed to hold the head as low as possible with the suspected antrum uppermost, for by this means the discharge may enter the meatus and be visible on rhinoscopic examination.

Transillumination.—This is carried out in a darkened room by placing a 10-volt electric lamp in the patient's mouth, and then comparing the degree of illumination of the two sides of the face, particularly the upper regions of the cheeks immediately below the eyelids (Plate I.). The diseased side will be dark, or relatively darker than the healthy side. A dental plate must always be removed from the upper jaw before using the test, because its presence would entirely prevent transillumination. This test is of great presumptive value, but only when taken in conjunction with other symptoms; for it must be remembered that an opaque antrum does not necessarily signify empyema, because a similar appearance may be caused by a deviated septum, excessive thickness of the antral wall, or a malignant growth within the antrum.

If, after the above tests, there be any doubt as to the presence of an empyema, the question can be definitely settled by exploration.

Exploration.—The outer wall of the inferior nasal meatus is rendered anaesthetic with cocaine, and a suitable trocar and cannula, passed backwards and outwards between the anterior end of the inferior turbinal and the outer wall of the nose, are made to pierce the inner antral wall (Fig. 8). The trocar is withdrawn, and to the proximal end of the retained cannula a syringe is fitted, which is filled with warm boracic lotion or normal salt solution. The antrum is now washed out. If it contain any pus, this will be seen in the fluid which returns through the nostril.

After a little practice, intranasal exploration of the antrum is an easy, safe, and reliable procedure ; it is infinitely preferable to the removal of what may prove to have been a valuable tooth.

Skiagraphy.—If a good x-ray negative of the face be made, an antrum containing pus will exhibit a blurred indefinite outline when compared with the normal sinus of the healthy side.

Prognosis.—An empyema of dental origin is more favourable than one of intranasal origin caused by the specific organisms of one of the acute infectious diseases. The prognosis is more favourable when the corresponding nasal cavity is free from conditions which hinder drainage, such as polypi and septal deviations. When other sinuses are simultaneously diseased, it may be necessary to cure them before the antrum will improve. Excesses in alcohol and tobacco are detrimental to rapid cure of an empyema.

Treatment.—One of two methods are generally adopted: (1) alveolar drainage, or (2) intranasal drainage.

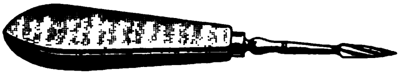

FIG. 9.—Antral perforator.

(1) Alveolar Drainage.—Under nitrous oxide anaesthesia the second bicuspid or one of the molar teeth is extracted, the buccal socket perforated, and a suitable plug or tube inserted. Irrigation with warm normal saline solution, concentrated boracic lotion, or some simple antiseptic should be practised twice daily at first, and then with diminishing frequency as the discharge lessens. When no pus is washed out after an interval of a week since the previous irrigation, the case may be considered cured and the plug removed. The alveolar method is useful in a simple, uncomplicated chronic empyema of dental origin, for even if it fails to cure, the discharge will always be lessened and its fetor diminished. It may be employed in all recent cases, that is, those in which the symptoms have only lasted for months rather

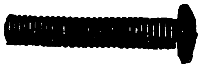

FIG. 10. — Vulcanite antral plug (twice the ordinary thickness used by the author).

than years, and occasionally it will cure an abscess of much longer duration. Alveolar puncture and drainage have advantages for the very old, those in broken health, the nervous, and for the busy man who cannot afford the time for a major operation which will demand, at least, a fortnight's rest from work.

(2) Intranasal drainage is called for (*a*) in cases in which alveolar drainage has failed to cure the discharge; (*b*) when the disease is of intranasal origin and no carious teeth are present; and (*c*) when polypi or degenerate mucous membrane are present in the middle meatus, for in these circumstances it may be assumed that the antral mucosa is similarly diseased, and simple alveolar irrigation will not effect a cure.

The drainage may be secured by one of two methods: (*A*) The Intranasal route.—Under general anaesthesia the anterior half of the inferior turbinal is removed by means of scissors and a wire snare, so that the nasal aspect of the inner antral wall is exposed. A hole the size of a sixpence should now be made through it by means of suitable burrs, curved knives, forceps, or chisels; this opening must easily admit the first joint of the little finger, because intranasal wounds are very apt to contract. The after-treatment consists in irrigation of the antrum with mild antiseptics through the artificial opening; this must be continued with diminishing frequency as the discharge shews signs of ceasing. (*B*) An opening in the canine fossa with a counter-opening into the nose. —Under general anaesthesia the anterior half of the inferior turbinal is removed, and the posterior choana plugged with an aseptic marine sponge. An incision is then made over the canine fossa in the gingivolabial groove, the soft parts and periosteum turned up, and the canine fossa freely opened with mallet and chisel. The antral cavity is now carefully inspected, and *only the diseased mucous membrane* curetted away. By means of suitable forceps the inner antral wall is next removed, as well as any suppurating ethmoidal or maxillo-ethmoidal cells which may be found. Finally, when the oozing of blood has been checked by means of sterilised strips of gauze, the bucco-antral wound is drawn together by two or three interrupted horse-hair sutures. The after-treatment consists in intranasal irrigation until the healing process is complete; this should take about a month.

Both these operations give satisfactory results, but I prefer the route through the canine fossa, because it enables the surgeon to see the extent of the disease, and to deal with it alone without damaging healthy mucous membrane.

CYSTIC DISEASE OF THE ANTRUM.—This is met with in two forms: (1) cysts due to degeneration of a mucous gland of the lining membrane; this is a very rare condition; (2) dental cysts, which invade and may distend the antral cavity; this is the more frequent form of antral cyst.

Symptoms.—There may be slight pain in and around the cheek, but it is the recognition of a swelling under the cheek which usually alarms the patient. The external deformity may be extremely obvious, and the expansion of the inner antral wall may cause a narrowing of the nasal cavity with obstruction of nasal respiration. "Egg-shell crackling" is often well marked in that portion of the tumour which lies over the anterior wall of the sinus. Transillumination may shew that the distended sinus is lighter than the normal antrum. The contents of such cysts are usually viscous, yellowish-green, non-fetid, and may shew cholesterin crystals in great numbers.

Diagnosis.—A large dental cyst expanding the antrum is very likely to be confused with malignant disease of the upper jaw, but the diagnosis can usually be made by attention to the following points:—

Cyst.	*Malignant Disease.*
Pain slight or absent.	Severe pain of a neuralgic type.
Transillumination more or less clear.	Opaque on transillumination.
Fluid contents on exploration.	No discharge, and probably only a few drops of blood.

Treatment.—The most speedy and certain method of cure is to remove the whole of the cyst wall by an operation similar to that advised for the cure of chronic empyema of the antrum; the inner antral wall should also be removed, and free nasal drainage thus provided; the bucco-antral wound should be sutured at the close of the operation.

INFLAMMATION OF THE ETHMOIDAL CELLS (ETHMOIDITIS).—In considering the inflammatory changes which may be seen in the ethmoid bone, it will be convenient to discuss them as they affect the "lateral mass" of cells or the middle turbinal. Inflammation of the ethmoid may be acute or chronic, and it may or may not be associated with suppuration. The demands of space will only allow the consideration of one pathological condition, namely, chronic suppurative ethmoiditis. It should, however, be stated that chronic non-suppurative ethmoiditis may take the form of sclerosing or of rarefying osteitis; these changes are secondary to chronic inflammatory changes in the muco-periosteum which sometimes give rise to nasal polypi.

Chronic Ethmoidal Suppuration.—*Etiology.*—The factors leading to chronic suppuration in the ethmoidal cells have already been referred to, and a glance at the complicated galleries of cells composing the "lateral mass" shew how suitable are the conditions for the retention of inflammatory products and the spread of infective processes within them.

Morbid Anatomy and Pathology.—Clinical experience has amply proved that suppuration may not only be confined to the anterior or posterior group of cells, but also to certain cells of these groups. Extensive suppuration of the lateral mass is nearly always associated with suppuration in one or more of the neighbouring sinuses, antral, frontal, or sphenoidal. Rhinoscopic examination of the ethmoidal region will generally reveal the presence of a purulent discharge in the middle meatus, and the mucosa lining this region may exhibit various degrees of inflammation, from simple hyperaemia and swelling to extensive oedema and polypoid degeneration. If the same regions are examined with a blunt surgical probe, spicules of carious bone may be detected; but necrosis, in its ordinary sense, is almost unknown except as the result of tertiary syphilis.

Symptoms.—Pain is not, as a rule, severe so long as the drainage of the pus is free, but retention of inflammatory products may cause pain and discomfort over the bridge of the nose and the lower part of the forehead, and deep-seated pain in the orbit; pain may be most severe in the suboccipital region. There may be various degrees of nasal obstruction; this may be extremely pronounced when the suppurative inflammation is confined to the cell or cells in the anterior end of the middle turbinal. The discharge is usually free from fetor, and has a peculiar tendency to dry into shell-like crusts, which cling to the regions from which it is excreted. The pus may be seen in the anterior part of the middle meatus when the anterior cells are affected, or between the middle turbinal and the septum, or covering the posterior ends of the middle and inferior turbinals when the posterior cells are diseased. The sense of smell is frequently impaired or totally destroyed. Loss of memory, mental depression, inability to concentrate the attention, and general apathy—a condition designated as aprosexia—have often been noted in chronic ethmoidal suppuration.

The *diagnosis* will be arrived at by a consideration of the symptoms, coupled with a careful examination of the ethmoidal region. A unilateral, non-fetid crust-formation, limited to the middle meatus, should always arouse suspicion of suppurative ethmoiditis, and if these regions be examined with an ordinary probe, they will be found to be more sensitive and friable than on the healthy side. In many cases small areas of caries can be thus detected, and when this is so, the middle meatus is generally occupied by small bunches of prominent granulation-tissue. If the pus escape freely into the nose, no external evidences of the disease are seen, and the condition is spoken of as a "latent empyema"; when retention of inflammatory products occurs, there may be definite external swelling and fluctuation above the internal canthus.

Prognosis.—In addition to the symptoms already described, chronic ethmoidal suppuration carries with it the risk of meningeal complications, of which a considerable number of fatal cases have been reported.

Treatment.—Chronic suppuration confined to the cell within the anterior end of the middle turbinal calls for removal of that structure by scissors and snare. The same procedure must be adopted as a preliminary measure when the suppuration occurs in the lateral mass, for only by such means will free drainage be established and access to the lateral cells secured. If suppuration be limited to one or more of the anterior cells they should be anaesthetised with a 15 per cent solution of cocaine, and polypoid or oedematous granulations removed by suitable forceps, such as Luc's or Grünwald's ethmoidal forceps. The bony walls of the diseased cells can best be opened with suitable hooks, such as those introduced by Hajek for breaking down the front wall of the sphenoidal sinus. The patient must be kept at rest for forty-eight hours, and the nostril lightly packed with sterilised wool. When extensive ethmoidal disease is present, the patient should be anaesthetised, and the diseased areas freely removed with a sharp ring-curette or ethmoidal forceps until healthy, firm bone is reached. If an external swelling or fistula be present an attempt may be made to relieve it by the intranasal method, but failing to afford drainage by this route an external operation must be performed. Space will not permit a description of surgical details, but it will suffice to state that a free incision over the external swelling (Fig. 13) will be required, and the soft parts must be retracted so that the diseased ethmoidal cells may be exposed and efficiently removed with suitable curettes or sharp spoons. By such means free intranasal drainage may be secured and the external wound immediately sutured. In other circumstances, for example an orbital abscess of ethmoidal origin, it might be well to leave the external wound open and to employ a drainage tube for a few days until all acute symptoms have subsided, and then allow the skin incision to close.

INFLAMMATION OF THE FRONTAL SINUS may be acute or chronic. Of all the specific diseases influenza is the most prone to affect the frontal sinus. There can be no doubt that many cases recover spontaneously; thus, during the epidemic of December 1907, I saw four cases out of seven recover without any local treatment; this tendency to resolution is probably due to the favourable situation of the fronto-nasal canal for purposes of drainage.

Acute Inflammation.—*Symptoms.*—In addition to the symptoms of the general disease, of which the sinus inflammation is a complication, there is acute supraorbital headache, which may spread into the cheek, temple, ear, or cause intense pain in the eyeball or back of the eye. Such pains are much aggravated by coughing, straining, blowing the nose, and so forth. Pressure on the anterior wall of the sinus is painful, and if applied upwards and inwards on the floor of the sinus may be unbearable. If the fronto-nasal canal become blocked, so that the inflammatory products cannot escape, the external soft tissues covering the sinus become red, swollen, and oedematous, and in these changes the upper eyelid may take part, so that the patient cannot open his eye. If the tension be not relieved by nature or art, pus will make its way through the thinnest part of the sinus wall beneath the inner end of the eyebrow, and fluctuation may then be detected. The eyeball is rarely displaced in acute inflammation of the frontal sinus. Examination of the nasal cavity will reveal swelling and congestion of the nasal mucosa, and probably a purulent discharge in the anterior region of the middle meatus.

Diagnosis.—In addition to the above symptoms, a rhinoscopic examination should be made, and any discharge removed from the middle meatus; this area should then be painted with a 10 per cent solution of cocaine.

If a suitably curved probe can then be passed into the frontal sinus, and its removal be followed by a discharge of pus, the diagnosis will be clear. I have twice seen acute frontal sinus inflammation mistaken for erysipelas, and frequently known it diagnosed as acute neuralgia. These mistakes might be avoided if the nasal cavities were more frequently examined.

Prognosis.—Many cases recover without treatment, and few would pass on to the chronic stage if efficient local treatment were undertaken in the early stages of the sinus inflammation.

Treatment.—In addition to the general treatment of the constitutional disease, the aim of local treatment should be to secure free, unhindered, spontaneous drainage from the inflamed sinus. To this end a 20 per cent solution of cocaine may be applied on a wool-mop to the anterior parts of the middle meatus, in order to cause contraction of the engorged tissues around the lower part of the fronto-nasal canal. Scarification of this region would tend to effect the same purpose. In the absence of the medical attendant the patient should inhale, every half hour or hour, mentholised steam (ʒss. of 30 per cent alcoholic solution of menthol to a pint of boiling water). The patient's sufferings may sometimes be considerably shortened if the inflammatory products can be washed out of the sinus by the passage of a suitably curved cannula, but the necessary manipulations require expert aid. Considerable relief may often be obtained from hot or cold applications to the forehead, and from the internal administration of aspirin, phenacetin, or antipyrin. When these measures fail it may be assumed that pus is retained under tension, and an external operation should at once be performed.

Operation.—The skin of the forehead and eyebrow should be rendered aseptic, and under general anaesthesia a curved incision is made immediately below the inner half of the eyebrow, curving downwards and inwards to terminate just above the internal canthus (Fig. 11). The soft parts and periosteum are retracted, and the lower, inner, and anterior sinus wall is thus exposed; a small disc of bone should now be removed with chisel and mallet, and the inflammatory contents gently syringed out of the sinus with sterilised water or warm perchloride of mercury lotion (1 in 3000). Every effort should be made not to damage the mucous membrane. The fronto-nasal canal may now be explored with a blunt probe, and its passage rendered free. A drainage tube is passed into the sinus through the opening in the bone, the extremities of the skin wound united with horse-hair sutures, and a hot fomentation applied. During the following days the sinus should be gently irrigated with a warm mild antiseptic lotion and the external dressings replaced. After ten days or a fortnight, when inflammatory symptoms have subsided and the sinus has ceased to secrete pus, the external wound may be allowed to close.

Chronic Inflammation of the Frontal Sinus.—Chronic suppuration of the frontal sinus is almost always associated with similar changes in at least some of the anterior ethmoidal cells; oftentimes the whole ethmoidal labyrinth (anterior and posterior cells) is diseased.

Morbid Anatomy and Pathology.—The changes in the mucous membrane are identical with those described in the case of the antrum. In long-standing cases the posterior wall may be eroded, so that the dura mater is exposed, or the septum separating the right and left sinuses may be absorbed. With obstruction of the fronto-nasal canal, even though not complete, the floor of the sinus may be gradually forced downwards, so that the eye is displaced downwards and outwards. The bony wall may eventually perforate and give rise to a subcutaneous abscess; the perforations most frequently occur below the inner end of the eyebrow. The intranasal appearances are practically identical with those seen in chronic ethmoidal suppuration; in fact the latter condition nearly always exists in a greater or less degree. The affection is frequently bilateral.

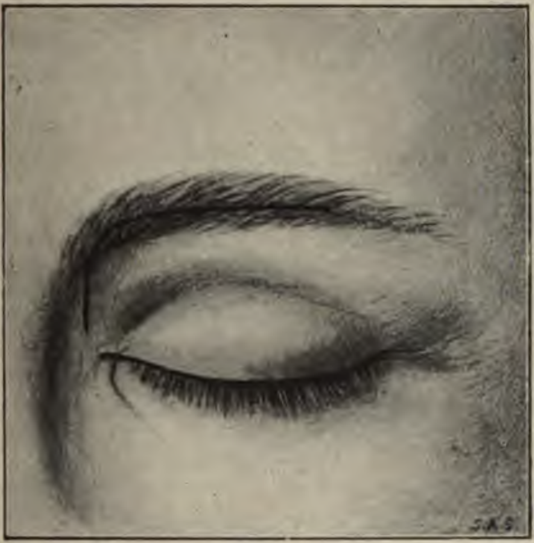

FIG. 11.—To shew line of incision for opening the frontal sinus in acute inflammation.

Symptoms.—There is often only a purulent discharge from the nose, but pain and nasal obstruction may also be present. The discharge is usually bright yellow, and may be very fetid; it is seen in the anterior part of the middle meatus, and may be blown forwards into the handkerchief; if obstructive lesions constrain the pus to flow backwards the patient may complain of "post-nasal catarrh," "pharyngitis," or other throat symptoms. The pain may be slight or severe, according to the degree of tension within the sinus, and hence an acute cold in the head generally aggravates the pain by causing swelling of the mucosa which hinders free drainage. Firm pressure directed upwards and inwards against the floor of the sinus will nearly always elicit pain. As a result of the chronic discharge of pus from the sinus, polypi and catarrhal changes in the nasal mucous membrane are frequent, and hence the patient is subject to "colds in the head." For similar reasons the sense of smell is often blunted.

Diagnosis.—In frontal sinus suppuration the discharge is apt to flow from the anterior part of the middle meatus, whereas in antral empyema the pus is seen farther back between the middle and inferior turbinals. To make certain that the frontal sinus is the source of the pus, the middle meatus should be carefully cleansed and anaesthetised with a 10 per cent solution of cocaine. An attempt should then be made to pass a fine, suitably curved cannula into the sinus, and by forcing air into it to drive out the discharge into the middle meatus, where it can be observed by the surgeon. A successful skiagraphic negative of the frontal regions will shew the outline of the diseased sinus much less clearly than that of the healthy one. Transillumination of the frontal sinus is, in my experience, of little value in determining the presence or absence of pus. The difficulties of diagnosis will be much enhanced when there is simultaneous disease of the neighbouring sinuses.

Prognosis.—Uncomplicated chronic suppuration of a frontal sinus with free intranasal drainage is rarely dangerous to life. The gravity of the case will be in direct proportion to the amount of coexistent ethmoidal disease. The following circumstances render operation advisable: (1) the presence of constant or intermittent pain; (2) coexistence of extensive ethmoidal disease; (3) a state of bad general health, especially when this can be fairly attributed to disorders of digestion induced by swallowing purulent discharges; (4) general mental depression; (5) orbital displacement, recurrent attacks of oedema of the upper eyelid, and external fistula.

Treatment.—Two methods may be adopted, the intranasal and external.

(I.) Intranasal treatment consists in removal of the anterior half of the middle turbinal, and of any polypi, granulations, or carious bone in the anterior ethmoidal region—in fact, the establishment of free drainage from the sinus, which should then be irrigated daily with warm solution of peroxide of hydrogen, weak carbolic, boracic, or similar lotions. Such a method may be expected to succeed in a small percentage of cases, in which the sinus possesses an even contour, drains freely into the nose, and in which the adjacent ethmoidal cells are fairly healthy. Only rarely can patients be taught to carry out the manipulations themselves, and even then a fresh access of "cold" is apt to be followed by return of the suppuration.

(II.) External operation.—The extent and nature of this will vary with the amount of coexisting ethmoidal disease. (*A*) When the ethmoidal disease is slight and limited to the anterior cells, the following method may be adopted. The preliminaries and incision are identical with those described for acute suppuration, except that, in addition, the posterior choana should be occluded with a sponge. Having opened the sinus, its whole anterior wall must be removed with suitable bone-forceps (Fig. 12), the diseased mucous membrane entirely curetted away from every corner and recess in the sinus, and the fronto-nasal canal enlarged by suitable burrs. The bony wound is then lightly packed with gauze, and an external dressing applied. The cavity of the sinus must be packed daily for three or four weeks until it has become entirely obliterated by granulation-tissue, for only by this means can a radical cure be guaranteed. A sinus of average size will take from four to six weeks to heal completely. If the operation be skilfully performed and the after-treatment carefully carried out, it is surprising how little deformity results in many cases.

(*B*) When extensive ethmoidal disease is present with frontal sinus suppuration. In these circumstances Killian's operation is eminently satisfactory (Fig. 13). The incision already described is prolonged downwards in front of the internal canthus, and then slightly outwards on the ascending process of the superior maxillary bone (Fig. 13). After separation of the soft parts the anterior wall of the sinus is completely removed to the level of the supraorbital ridge, as is also the floor of the sinus, so that the orbital fat rises into the lower part of the cavity. A second opening is now made by removing a portion of the upper end of the nasal process of the superior maxillary, which closes in the anterior part of the lateral mass of the ethmoid. By this means ready access to the ethmoidal cells is obtained, and diseased areas can be easily removed. At the close of the operation the external wound is completely sutured, and the bridge of bone, which is left between the two openings above described, prevents undue sinking in of the soft parts and consequent

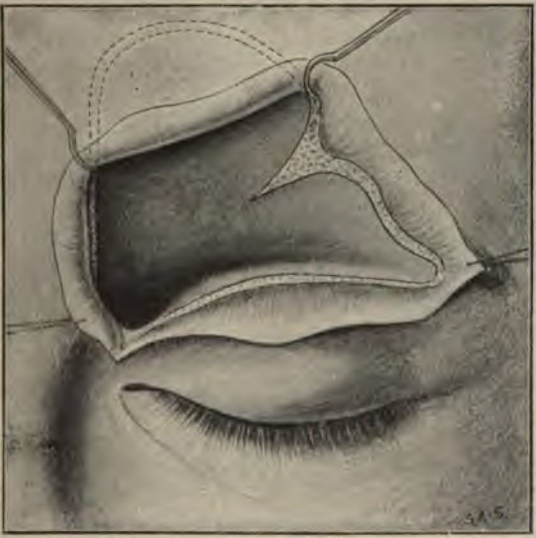

FIG. 12.—To shew complete removal of the anterior wall of the frontal sinus. The dotted line indicates the upper limit of the sinus.

deformity. The operation is extensive, and requires great care and detailed anatomical knowledge of the parts which are encroached on, but it gives most excellent results, and is invaluable in cases of extensive fronto-ethmoidal suppuration.

Complications of Radical Frontal Sinus Operations.—(*a*) Diplopia, due to interference with the superior oblique muscle; as a rule the double vision passes off in the course of two or three weeks. (*b*) Delayed healing of the external wound, with suppurating fistula or recrudescence of suppuration in the sinus. These two complications are generally due to infection of the floor of the sinus by a neighbouring ethmoidal cell. (*c*) Spreading infective osteomyelitis of the frontal bone. This is nearly always fatal, and is due to imperfect drainage of the sinus cavity. The actual focus of infection is usually a suppurating ethmoidal cell near the floor of the sinus, and hence the importance of dealing thoroughly with these cells when the sinus is opened, and of providing for free drainage either into the nose or to the exterior, during the healing of the wound. The supervention of the complication may be suspected when redness, oedema, and tenderness of the tissues in the neighbourhood of the sinus are seen. The patient may linger for many weeks, or even months, but eventually succumbs to suppurative meningitis. Very free removal of all inflamed bone around the sinus, so that the dura is exposed, is the only means of averting a fatal termination.

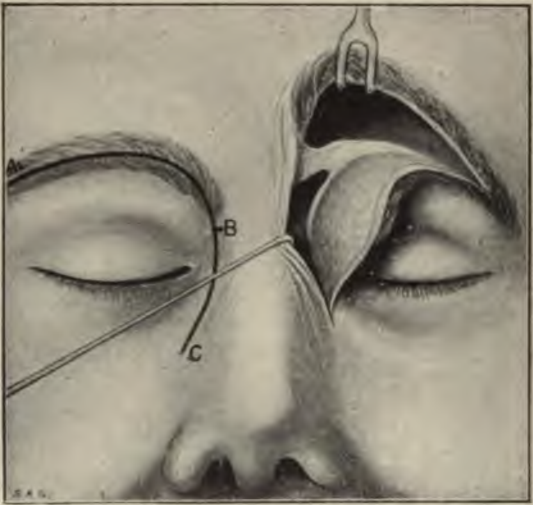

Fig. 13.—Killian's operation for chronic suppuration of the fronto-ethmoidal cells. ABC = Line of incision for Killian's operation; AB = Line of incision for radical operation on the frontal sinus when there is little ethmoidal suppuration; BC = Line of incision for external operation on anterior ethmoidal cells.

Inflammation of the Sphenoidal Sinus.—Chronic suppuration and its more acute exacerbations are the only forms of inflammation which need careful consideration; instances of acute inflammation of the sphenoidal sinus rarely come under notice.

Etiology.—Those factors which produce suppuration in other sinuses may induce a similar result in the sphenoidal cavity. It is often very difficult to ascertain whether the infection has been direct or has reached the sphenoidal sinus by way of the posterior ethmoidal cells, which are intimately connected with the outer half of the anterior wall of the sphenoidal air-cell. In my experience tertiary syphilis not uncommonly produces caries or necrosis of the anterior wall of the sphenoidal sinus and suppuration within the contained air-cell; similar lesions are uncommon in the frontal and ethmoidal sinuses.

Symptoms may be so slight as scarcely to attract the patient's attention. In other instances there may be pain, excessive post-nasal discharge, and disturbance of the senses of smell or of sight. When pus is retained within the sinus, or infection of the neighbouring veins and lymphatics occurs, grave intracranial symptoms may rapidly supervene. Pain may be mild or severe, and varies much in its situation. Occipital pain is said to be characteristic of sphenoidal sinus suppuration, but it is frequently frontal, temporal, or vertical, and I have recorded three cases in which deep-seated pain in the ear was proved to be due to the same cause. The discharge may pass unnoticed, or be the sole cause of complaint; it is frequently mistaken by patient and physician for "post-nasal catarrh," because of its tendency to pass backwards into the pharynx. In this situation it often forms tenacious crusts which are difficult to dislodge. The olfactory sense may be entirely destroyed, or "cacosmia," the sensation of an evil odour, may be a distressing symptom. Fever is usually absent, except when pus is retained under pressure, or grave meningeal complications are pending. Giddiness, mental irritability, depression, and apathy have been noted in other instances. The general health will suffer in proportion to the severity and frequency of the headaches, and to the amount of purulent discharge swallowed and absorbed into the system.

If infection spread beyond the bony walls of the sinus, either directly through abnormal dehiscences in its walls or as a result of caries, or by the lymphatics or veins, grave symptoms with serious consequences soon appear. Headache, rigors of a pyaemic character, vomiting, drowsiness, delirium, or coma would point to infective thrombosis of the cavernous sinus. Ocular symptoms usually develop on one side, and eventually appear on the other; as a rule they take the form of ocular paralyses, inflammatory changes in the fundus, chemosis of the conjunctiva, and proptosis. Exophthalmos, with sudden blindness of one eye, should suggest the spread of inflammation from the sphenoidal sinus. These symptoms often rapidly pass into or are masked by those of basal meningitis, to which sooner or later the patient almost invariably succumbs.

Prognosis.—The close proximity of the base of the brain and the cavernous sinuses to the sphenoidal sinuses render chronic suppuration within those air-spaces a serious condition. Even if the discharge be free and the natural ostium patent, clinical experience has shewn that fatal meningitis may supervene, and therefore, when once suppuration in the sphenoidal sinus has been diagnosed, the patient should be urged to submit to surgical treatment.

Diagnosis.—In a characteristic case anterior rhinoscopy would reveal a purulent discharge between the middle turbinal and the septum, with polypoid changes in the higher regions of the nose. Posterior rhinoscopy might be expected to shew pus discharging downwards over the posterior ends of the middle and inferior turbinals, with a tendency to form a viscid, adherent layer of mucopus over the lateral pharyngeal wall. To clinch the diagnosis the surgeon should endeavour to pass a fine cannula into the ostium of the sinus, and watch this orifice for the appearance of pus, when he injects air into the sinus. To carry out the required manipulations it may be necessary to remove the middle turbinal.

The main principle of **treatment** is to provide free, unhindered, spontaneous drainage from the sinus, and if this be secured, not only may grave danger be averted, but in many instances a cure of the suppuration may be attained.

Operation.—Under general anaesthesia the nostril is dilated with a speculum, the nasal cavity brightly illuminated, the middle turbinal bone completely removed, and the natural opening into the sinus is sought for; this may often be recognised by the pulsation of a drop of pus within its circumference. By means of suitable hooks and long, small, sharp spoons the anterior wall of the sinus and the neighbouring ethmoidal cells, which are usually suppurating, may be removed. No attempt should be made to curette the inside of the sinus, and still less to remove its lining mucous membrane. The cavity of the sinus is then carefully cleansed with some antiseptic, and gently packed with sterilised gauze. This should be removed in forty-eight hours' time, the sinus again cleansed, and once more repacked if this be necessary. Much care will be needed to prevent the opening into the sinus from becoming contracted by the growth of granulation-tissue, and frequent applications of lunar caustic or the galvano-cautery may be necessary. By such means suppuration may be entirely checked.

CHRONIC SUPPURATIVE PAN-SINUSITIS.—Frequently all the sinuses of one or both sides of the nose are in a state of chronic suppuration. The diagnosis and treatment of these cases need considerable experience, an intimate and practical knowledge of the anatomy of the nasal cavities, special technique, and often unlimited patience. Those who desire more detailed information on these matters must refer to special text-books on diseases of the nose, but practical knowledge can only be obtained by personal examination of many examples of the pathological conditions which have been briefly discussed.

HERBERT TILLEY.

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H. T.

4. DISEASES OF THE NASO-PHARYNGEAL CAVITY

Naso-Pharyngeal or Post-Nasal Catarrh

By Herbert Tilley, B.S., F.R.C.S.

By these terms is meant a morbid condition of the mucous membrane lining the post-nasal space, which causes an excessive discharge of mucus or mucopus. The symptoms may be acute or chronic.

Acute Post-Nasal Catarrh.—This is met with as part of the general catarrhal inflammation occurring in acute rhinitis, and their etiological factors are identical (*vide* Acute Rhinitis, p. 8). In addition to feelings of malaise and mild pyrexia the patient often complains of slight earache, deafness, and tinnitus, while the excessive discharge of mucus or mucopus produces additional discomforts. The treatment is practically identical with that of acute rhinitis.

Chronic post-nasal catarrh frequently accompanies the various forms of chronic rhinitis, and is practically due to the same causes, for example frequent attacks of acute rhinitis, different forms of nasal obstruction, fibrous remains of old adenoid growths, abuse of alcohol and tobacco, the "strumous" and gouty diatheses, and frequent or prolonged exposure to irritating atmospheres. In young adults the symptoms are very frequently caused by a mild form of chronic suppuration originating within the folds and recesses of the pharyngeal tonsil.

Morbid Anatomy and Pathology.—The mucous membrane lining the post-nasal space may be uniformly red and swollen, and the discharge from it may be excessive, viscid, and muco-purulent. In other instances the lining membrane is paler and shrunken, from atrophy of the glandular elements, and in these circumstances the secretion will be very sticky and tenacious. When the symptoms are caused by chronic inflammation in the pharyngeal tonsil, post-rhinoscopic examination may reveal a purulent discharge issuing from the lower part of the median cleft in the tonsil—the so-called Tornwaldt's disease (*vide* p. 18). A certain amount of chronic pharyngitis is usually associated with post-nasal catarrh.

Symptoms.—The main symptom is an excessive discharge from "the back of the nose," the removal of which may need constant hawking and sniffing. This is often worse in the morning, and retching or even vomiting may result from the efforts to dislodge the secretion accumulated during sleep. Slight deafness and tinnitus may be caused by the extension of the catarrh to the Eustachian tubes, whilst a certain amount of pharyngitis and laryngitis indicates the spread of the affection downwards Headache, mental apathy, and other reflex disturbances are not uncommon.

Diagnosis.—It must be remembered that post-nasal catarrh is only a morbid condition due to factors already enumerated, which must be carefully sought for. Special care should be taken not to overlook chronic suppuration of the nasal accessory sinuses, because a purulent discharge from those cavities is very liable to pass backwards into the naso-pharynx, and to be regarded as "post-nasal catarrh."

Prognosis.—When the symptoms are due to constitutional causes which render the mucous membranes liable to catarrhal inflammation, the prospects of cure are not so good as when certain local changes, capable of removal, are met with.

Treatment.—The causative factors already enumerated must be sought for and remedied, and especial stress must be laid on the remains of old adenoids, chronic rhinitis, nasal obstruction, and excesses in alcohol and tobacco. In a case of recent origin the nasal cavities should be cleansed night and morning with a warm alkaline antiseptic spray, *e.g.* ℞. Potass. chlorat., sod. bibor., sod. bicarb., sacch. alb. āā ʒss. Sig. ʒss. in half a tumblerful of warm water. In a more chronic case, with general thickening of the mucous membrane, and a tendency to the collection of viscid secretion, the nasal and naso-pharyngeal cavities should be cleansed with the preceding solution, and the post-nasal space then painted with nitrate of silver grs. x.-xx. ad ℥i., or grs. x. of iodine and iodide of potassium to an ounce of glycerin, or chloride of zinc grs. xx. ad aq. ℥i. Such applications may be made twice or thrice weekly according to the needs of the case. The remains of adenoids must be efficiently dealt with by surgical means, and the same may be said of the various forms of nasal obstruction. In all cases, mild as well as severe, tonics, change of air, attention to the digestion, and so forth, are invaluable aids in supplementing the more active local treatment outlined above.

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Tuberculosis may attack the naso-pharynx, giving rise in some cases to ulceration, and in others to a diffuse infiltration of the posterior aspect of the soft palate. Tubercle bacilli have been detected in adenoid vegetations; and there is good reason to believe that secondary infection of the cervical lymphatic glands is often due to this source.

Syphilis in all its forms has been observed in the naso-pharynx. Primary syphilis of the naso-pharynx is almost exclusively due to infection by means of the Eustachian catheter. Secondary syphilis occurs in connexion with a similar affection of the pharynx. It is not at all uncommon for tertiary ulceration of the naso-pharynx to occur quite independently of any mischief in the pharynx, hence the importance of a rhinoscopic examination in these cases. When such ulceration occurs simultaneously on the upper surface of the soft palate, the resulting cicatrisation may cause extensive adhesions between the soft palate and the posterior pharyngeal wall, so that the nasal cavities may be almost completely cut off from the oro-pharynx (*vide* also p. 137).

F. DE HAVILLAND HALL.

HYPERTROPHY OF THE PHARYNGEAL TONSIL

SYNONYMS.—*Adenoid Vegetations; Post-Nasal Growths.*

By Sir FELIX SEMON, K.C.V.O., M.D., F.R.C.P., and P. WATSON WILLIAMS, M.D.

The aggregation of lymphoid tissue on the roof and posterior wall of the naso-pharynx, known as the pharyngeal or Luschka's tonsil, is very similar in structure and formation to the faucial tonsils, and is liable to the same morbid changes. In fact, in about 50 per cent of the cases hypertrophy of the faucial tonsils and post-nasal adenoids coexist; but the latter disease gives rise to a distinctive group of symptoms, the clinical importance of which, first recognised and described by Wilhelm Meyer of Copenhagen in 1868, has become very generally appreciated.

The question is often asked whether adenoids are a comparatively "new" disease? Nothing could answer this question more effectively in the negative than the following reproduction (Fig. 14) of the portrait of the Emperor Ferdinand the First of Germany, King of Spain, which was painted in 1524 by the great Dutch painter, Lucas of Leyden.

It is in the Galeria degli Uffizi at Florence, and gives such a typical illustration of the disease, that from it alone the diagnosis of the affection can be made to-day, more than 380 years after its execution, with absolute certainty. The usually well-marked Habsburg family type has been completely lost through obstruction to nasal breathing continued for many years. The whole face has become elongated, the nose is pinched and much too small for the face, the nostrils are narrow, the upper lip retracted, the face shewing the expression of retarded mental and bodily development. Wilhelm Meyer, too, has drawn attention to several antique portraits, busts, and statues which render it extremely probable that the originals had suffered from adenoid vegetations.

Etiology.—Post-nasal adenoid hypertrophy is a disease of early childhood, a period when all the lymphatic structures are especially active. It has been observed as early as the tenth month; the symptoms generally date from birth or early infancy, becoming well marked, as the hypertrophy increases, by the fourth or fifth year, if not before. Thus there is little room for doubt that the affection is often congenital in origin. The majority of cases come under our notice between the ages of five and fifteen; and the adenoids, though sometimes persisting and still more rarely extending after the age of twenty, in the great majority of cases participate in the retrogressive changes and atrophy common to many lymphatic structures after the age of puberty. By this time,

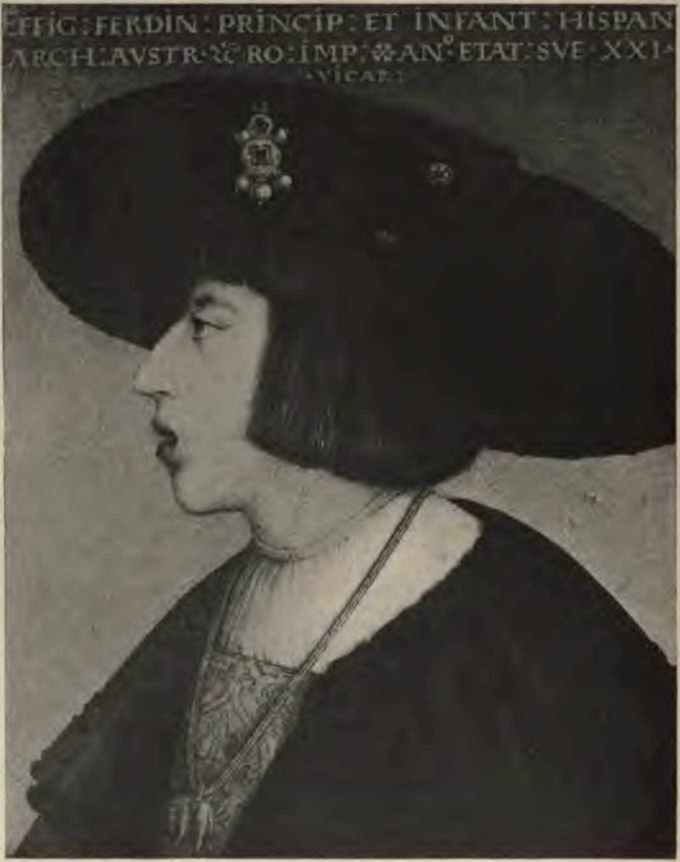
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FIG. 14.—Portrait, painted in 1524, shewing the adenoid facies.

however, the health and development of the patient are often permanently impaired.

The considerable influence of heredity in their occurrence is shewn by the frequency with which several members of a family suffer from adenoids. This influence is probably indirect, and is due to the transmission of the "strumous" diathesis in which we observe so marked a tendency to hypertrophy and degeneration of the lymphatic glands and a decided proclivity to tuberculous affections. Further, we often find, associated with the rhino-pharyngeal affection, various defects in development, such as a high-arched or V-shaped palate, contraction of the superior maxilla and consequent encroachment on the nasal fossae, and cleft palate. The importance of nasal stenosis as an etiological factor in producing the adenoid hypertrophy has been overestimated; nasal stenosis and chronic nasal catarrh usually are the results of post-nasal growths and concomitant defects in development.

A cold and damp climate disposes to the disease by increasing the tendency to catarrhal affections; and in warm and dry climates the disease is less common; thus, Massëi remarks that in Italy the disease is very rarely observed in any marked degree. Measles, scarlatina, and influenza are apparently very frequent exciting causes of the disease. On the other hand, it is equally certain that the presence of adenoids very greatly increases the risk of infection in various exanthems, and the liability to colds and bronchitis; thus it is often very difficult here to distinguish between cause and effect. Finally, in many patients who are otherwise healthy and strong we find no obvious cause for the glandular hypertrophy.

We have no certain knowledge of the physiological functions of the lymphoid tissue in the upper air-passages; but it is probable that they furnish leucocytes which are protective against the inspired micro-organisms that always exist in these parts. This question is more fully discussed in the section on the diseases of the tonsils. But it is quite certain that the pharyngeal tonsil, when in a condition of chronic hypertrophy and degeneration, like all tissues of low vitality, has lost its power of resisting the invasion of pathogenetic microbes, and is a ready portal of entrance for tubercle bacilli. Thus, the pharyngeal and particularly the cervical lymphatic glands are frequently affected and become enlarged in cases of adenoid vegetations, and one of us (F. S.) has twice seen retropharyngeal abscess associated with adenoids.

Pathology.—The growths occupy the vault and posterior wall of the rhino-pharynx, forming either a large cushion-like mass (see Fig. 2, Plate II.) or an aggregation of numerous large and irregular projections. They are covered with ciliated epithelium and the surface is coarsely lobular or mammillary. The substance of the growths consists of a connective-tissue reticulum filled with lymph corpuscles, the trabeculae being formed of branching corpuscles which have generally lost their nuclei. The tissue is, as already mentioned, very similar to that of the faucial tonsils, differing only in the absence of the crypts, the relatively small amount of connective tissue, the high vascularity, and the ciliated epithelium. Tuberculous tissue has been observed in the vegetations, and, very rarely, small cysts also. In adult patients we generally find the growths more or less atrophied, and firmer in texture from the preponderance of connective tissue.

The symptoms vary very much in kind and in severity : thus, whilst in some cases there is little to observe but nasal obstruction or deafness, in the vast majority the symptoms are so characteristic that the general aspect alone, as shewn in Fig. 14, is sufficient for the practised eye to make a diagnosis of post-nasal growths. The nose becomes pinched, the alae nasi fall in from long-continued disuse of the dilator muscles, and a dimple forms in the angle between the superior and inferior lateral cartilages. The upper lip is retracted, the upper incisors shew, the naso-labial fold is more or less obliterated, and, the inner canthus of the eye being drawn down, the eyelids droop and the whole face lengthens ; moreover, the necessity of breathing through the mouth gives an expression of dulness and vacuity which is still further increased by deafness. The child is generally pale and unhealthy-looking, and the cervical lymphatic glands are often enlarged.

Defective growth and all the evils due to mechanical obstruction to respiration in the young are often observed; as, for instance, chronic pharyngitis, colds in the head, laryngitis, and bronchitis. Dr. Eustace Smith observes that in childhood symmetrical retraction of the infra-mammary region and depression of the ensiform appendix (pigeon-breast) owe their origin with few exceptions to rhino-pharyngeal obstruction, the retraction of the chest-wall being directly due to pulmonary collapse. If this collapse be extensive, the lower part of the sternum becomes prominent from retraction of the cartilages of the ribs, whilst the recession of the infra-mammary and epigastric regions with each inspiration, noticed in very young children suffering from rhino-pharyngeal obstruction, results, if long continued, in permanent retraction of these parts. Moreover, in infants and young children with adenoids it is common to find collapse of the upper parts of the lungs ; and there may be deficient resonance with weak, harsh breathing in the supraspinous fossae, extending down to a short distance below the scapular spines : this may be accompanied by a dusky tint of the lips with other signs of imperfect aeration of the blood. Dr. Smith reminds us that, at this period of life, a high-pitched percussion note in the supraspinous fossae, without notable alteration in the breath-sounds, is commonly due to a patch of pulmonary collapse; and that when the rhino-pharynx is obstructed by a mass of adenoid growths, very hollow breathing, conducted from the pharynx, is heard over the upper part of the chest on both sides—a combination of physical signs which often leads to an erroneous diagnosis of serious disease.

The breathing of children suffering from advanced adenoid vegetations is peculiarly noisy and snuffling ; this is very noticeable during eating and drinking, and especially in sleep. Whereas in the daytime respiration is

PLATE II.

Fig. 1. The normal rhinoscopic image, shewing the Roman arch of the choanae narium. In children up to the age of puberty the normal small naso-pharyngeal tonsil is usually present in the vault of the naso-pharynx, but in health it never occludes any part of the choanae.

,, 2. Rhinoscopic appearance of a mass of adenoids concealing the upper part of the choanae.

,, 3. Abnormally large cushions of the Eustachian tube, a condition that should not be confounded with adenoids on digital exploration.

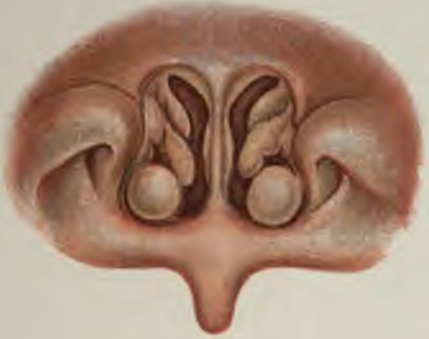

Fig. 1.

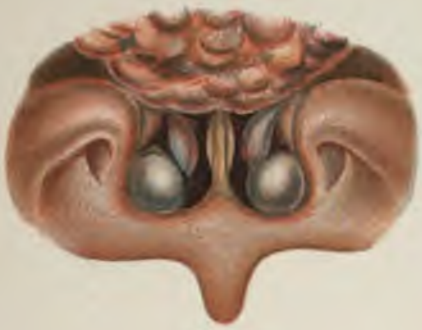

Fig. 2.

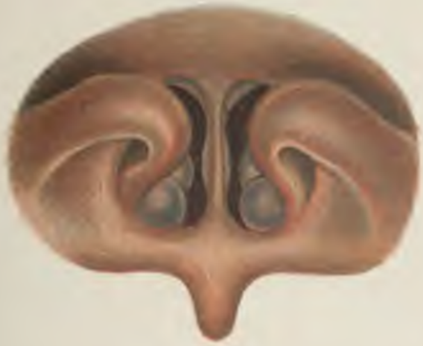

Fig. 3.

mainly by the mouth, the physiological habit of nasal respiration reasserts itself during sleep; moreover, the tongue tends to fall back against the soft palate, by which respiration is still further embarrassed, and snoring is set up. Suffocative "night-terrors" often occur; the little patients are restless in bed and their sleep is much disturbed: for as the embarrassed respiration through the nose creates an excess of carbonic acid gas in the blood, the *besoin de respirer* arouses the child so far as to take a few breaths by the mouth.

Speech is affected as the nasal obstruction interferes with the pronunciation of certain consonants: thus, B is substituted for M, D for N, G for NG and K, F for TH, and so forth. The voice is also remarkably toneless and flat, since the rhino-pharynx, being occupied by the growths, loses its resonant functions. Apart from these effects of nasal obstruction the children are usually backward in learning to speak and read; and articulation is very often defective, partly from want of tone in the palate muscles, and partly from deafness. To the deficient aeration of the blood must further be ascribed the lassitude, the "ready flagging," the headaches and giddiness of the little patients, and their inability to fix their attention (Guye's "aprosexia").

A peculiar harsh, dry, barking, reflex cough, independent of any bronchitic affection, is a very frequent complaint; it is usually worse at night. Cough is further induced by the accumulation of mucus in the back of the throat trickling down to the larynx, or by catarrhal affections of the upper respiratory tract. The soft vascular adenoids bleed so readily that the secretion is often blood-stained; and blood, even in considerable quantities, may be coughed up or, passing into the stomach, may be vomited. Whilst a history of inveterate cold-taking with constant running from the nose is usual, yet, on the contrary, the complaint may be that the child has a particularly dry nose, and that he never uses a pocket-handkerchief; although his speech sounds as though he has a cold in his head. Asthma, stuttering and stammering, laryngismus stridulus, chorea, nocturnal enuresis, and even convulsions and epilepsy, are among the neuroses that have been attributed to the presence of adenoids. We would, however, particularly warn the reader against too readily interpreting a coexistence of adenoids with any of these affections in the light of cause and effect.

Deafness in greater or less degree—sometimes periodical and coincident with cold in the head, sometimes constant—is one of the most frequent complications of adenoids, although very often treated lightly by the parents, who trust that the child will grow out of it; or they regard it as mere "inattentiveness." From the gradual absorption of the air in the middle ear which cannot be renewed—either in consequence of the Eustachian tubes becoming obstructed by catarrh in the naso-pharynx, or from paresis and interference with the action of the levator palati and salpingo-pharyngeus muscles—the tympanic membranes become much retracted. The membrane is often thickened and somewhat opaque and congested. From retention of the catarrhal secretions otitis media purulenta may arise with subsequent perforation, otorrhoea, and granulations. The extreme degrees of depression of the drum-heads are practically never seen in children except in connexion with adenoid vegetations. Should a child suffering from adenoids be attacked by scarlet fever or diphtheria, ear-complications, often severe and even incurable, are almost the rule.

In adults, the growths having usually become more or less atrophied whilst the rhino-pharyngeal space has increased, nasal obstruction and mouth-breathing generally disappear; though many of the evil effects persist.

On examining the fauces the soft palate may be seen to be relaxed, and its distance from the posterior wall of the pharynx unusually great. If the tonsils are not greatly hypertrophied, as in these cases they often are, numerous enlarged follicles on the posterior wall of the pharynx may be seen, unless they are obscured by the muco-purulent secretion descending from the rhino-pharynx. The growths themselves may be examined by rhinoscopic inspection and by palpation. Even in very young children it is occasionally possible to obtain a view of the rhino-pharynx with the rhinoscope. The growths appear either as a greyish-pink, gelatinous, cushion-like mass with vertical ridges and furrows, or as an aggregation of stalactite-like projections crowded together and presenting an irregular mammillated surface growing from the vault and posterior wall (Fig. 2, Plate II.). They often extend to the fossae of Rosenmüller, or occlude the orifices of the Eustachian tubes more or less, sometimes forming adhesions with the posterior lips. The Roman arch formed by the upper insertion of the vomer into the roof of the naso-pharyngeal cavity and the choanae are partially shut off from view; or the whole rhino-pharyngeal space may be filled with masses of growth. The surface of the mass is often more or less covered by viscid muco-purulent secretion. In adult patients it is not difficult to make the rhinoscopic examination.

For digital exploration of the rhino-pharynx the child is seated in a chair, the physician standing on the right side and holding the head firmly with the left hand, the right forefinger, protected by a finger-guard, or by a napkin, or by a cork between the patient's teeth, is rapidly passed behind the posterior pillars of the fauces, and thence upwards to the roof of the rhino-pharynx, and swept rapidly over the whole of the post-nasal space so as to determine the size, consistency, and location of the vegetations. As the forefinger impinges on the soft adenoids, the sensation reminds one of a bag of worms. However gently and carefully the examination be made, there is almost always some bleeding, and on withdrawing the finger it is stained with blood. Disagreeable though the digital exploration be, we must not be deterred from employing it, unless the posterior rhinoscopic examination yields absolutely satisfactory results; indeed, in the opinion of one of us (F. S.), palpation is superior to the latter method in enabling us to form a definite notion of the quantity of the growths present.

In the affection designated, for want of a better and more definite term, the status lymphaticus, the lymphoid tissues throughout the body are enlarged, and in this hypertrophy the tonsils and other lymphoid aggregations in the pharynx and post-nasal space participate, tending to result in the usual symptoms of adenoids, but in conjunction with the exceedingly dangerous constitutional condition inseparable from the status lymphaticus. Needless to say, the suspicion of the existence of this hitherto ill-defined disease should make the medical attendant exceedingly cautious in recommending operative measures which are not absolutely necessary, as it adds immensely to the danger. (See art. "Status Lymphaticus," Vol. IV. Pt. I. p. 482.)

Diagnosis.—It is only in infants or very young children, whose undeveloped features do not shew the characteristic facial aspect described above, that a difficulty in diagnosis should be possible. Nasal discharge and snuffling respiration, which as we have seen are marked symptoms in adenoid cases, are also frequently associated with congenital syphilis. But in syphilitic infants the nostrils are dry and shew radiating linear fissures; and, nasal obstruction being more complete, they are unable to take the breast. Moreover, other signs of the constitutional disease are usually present. Other kinds of growth in this region are extremely rare in children. In adults a differential diagnosis may have to be made between persistent adenoids, fibroma, nasal polypi extending backwards from the nose, and moriform hypertrophy of the inferior turbinals; all of which, with the exception of nasal polypus, are rare conditions, and may readily be distinguished by the seat of origin, colour, or consistency. In making a digital exploration, hypertrophy of the Eustachian cushions (see Fig. 3, Plate II.) should not be mistaken for adenoids.

Prognosis.—The prognosis is very favourable on the whole, provided no serious complications have arisen; broadly speaking, it stands in direct proportion to the patient's age and to the length of time the obstruction has existed. The most brilliant results are obtained by timely operation in young children; but the prospect, of course, is less favourable if organic changes have once taken place in the middle ear, or thoracic deformities are definitely established, or the time has passed when, by relief of the obstruction, an advantageous change could be expected in the configuration of the face. Although the adenoid growths, as a rule, atrophy spontaneously after puberty, and, with the increasing size of the rhino-pharyngeal space, the symptoms usually disappear, yet, before that age is attained, not only does a child run great risks of permanent deafness and impaired health and development, but it is also constantly exposed to attacks of catarrh and bronchitis, and is increasingly liable to contract the various exanthems. Moreover, in a certain number of cases the spontaneous atrophy is very partial; in others the symptoms do not vanish with the disappearance of the growths; whereas, by timely operation the whole disease can be completely and permanently eradicated and all these risks to health removed. Children almost invariably shew a most remarkable improvement in general health and intellectual development within a short time of the operation; instead of the pale and dusky complexion and of the dull woe-begone expression, we are greeted by brightness and intelligence, healthy respiration dilates the lungs, the chest develops, the deafness disappears and the patient increases in stature, weight, and activity. In short, removal of adenoids in really suitable cases is one of the greatest medical blessings of our era, and must have a far-reaching effect upon the health of future generations.

Our advice should be as follows:—If the patient be under twelve, and if any or several of the classical symptoms be well marked, whilst certainly admitting that the child may escape all the dangers involved in the disease, yet all the disadvantages of postponement may be removed by an operation which, if properly and skilfully performed, is practically devoid of danger. Of course no unnecessary operations should be performed, but in doubtful cases it is better to operate. We must not definitely promise that the growths will not recur, for even after very thorough and complete extirpation recurrence takes place in a small percentage (in our experience amounting to 1 per cent), especially after influenza. Moreover, some of the symptoms, especially speech defects, may persist for a considerable time after treatment. In no circumstances should a disappearance be promised of any of the "reflex" neuroses, which have been attributed to adenoids, such as asthma, stammering, enuresis, epilepsy.

Treatment.—Unless the vegetations be small, and not productive of any of the more serious symptoms above enumerated, no time should be lost in internal medication or change of air. Whilst again deprecating unnecessary operative interference, one of us (F. S.) must confess that more than once in the light of subsequent events he has regretted that, guided by the wish to spare the patient an operation which at the time did not seem to be urgently required, he had not laid more emphasis on the risks of delay. Moreover, there cannot be the least doubt that the operation itself acts indirectly as a powerful tonic, and promotes the desired restoration to health more effectually than any amount of cod-liver oil, extract of malt, and iodide of iron.

Operative treatment is called for in the great majority of cases presenting definite symptoms, and the younger the child the greater the reason for removing the growths without delay. For their complete extirpation a general anaesthetic ought to be employed, at any rate in children; and, though in adults removal may be done under nitrous oxide gas, or even with local anaesthesia by cocaine or novocaine, a general anaesthetic is desirable. Although the operation is frequently—on the Continent generally—performed under a local or very short-lived general anaesthetic, one of us (F. S.) still, for reasons of thoroughness, prefers chloroform, believing that it is quite safe, if administered by skilled hands, slowly and cautiously, and not pushed to the abolition of the cough-reflex, which protects the larynx against the entry of blood. Fortunately this reflex is the last to go (Semon and Horsley); and the administration of the anaesthetic should, therefore, cease as soon as the conjunctiva is insensitive. One advantage of chloroform over gas or ethyl-chloride is that the latter gives a very short time for operating. It is very possible that in some cases of death under the anaesthetic in operation for adenoid growths, the real cause of the fatal result was an unrecognised *status lymphaticus.* Gas or ethyl-chloride or ether followed by chloroform, is the usual anaesthetic employed by one of us (W. W.); ether alone is not well borne by young children prone to bronchitis. It is possible to remove both tonsils and adenoid growths under nitrous oxide gas alone; but, in our opinion, there is less opportunity for a complete removal of the growths, and therefore a greater likelihood of recurrence, than when the operation is somewhat more deliberately performed under a more prolonged anaesthesia; and whenever operative interference is undertaken, the importance of a thorough and radical removal cannot be overestimated.

As regards the particular method of removing the growths there is wide choice. Some operate by scraping with the finger-nail, others by curetting with post-nasal cutting curettes introduced through the mouth; or with a straight curette as employed by Meyer through the anterior nares; or by the use of cutting forceps, such as Loewenberg's; or by snaring with the cold or galvano-caustic wire, and destruction by caustics or the galvano-cautery. Each of these methods has its advocates, nor can the surgeon confine his practice to any one method.

Our own practice is to have the patient lying on his back with the head well extended and low down, a small pillow being placed under the neck. The mouth being kept open by a gag on the left side, held by an assistant, the operator, standing on the patient's right, passes a Gottstein's curette or one of its modifications (St. Clair Thomson, Delstanche, or Bechstein) behind the soft palate to the vault of the pharynx close to the posterior border of the septum, and then while gently but firmly pressed against the posterior wall it is drawn down so as to cut away the whole mass of growth. If vegetations are situated laterally in Rosenmüller's fossae, these are removed in a similar manner with Hartmann's curette. The right forefinger is then introduced, and rapidly swept over the vault into the fossae of Rosenmüller and over the Eustachian orifices, ascertaining whether anything has been left behind, and scraping away, if necessary, any remnants of growth with the finger-nail. The curette usually has to be introduced several times. Haemorrhage is always very free for a few minutes, but soon ceases spontaneously. Secondary haemorrhage is exceedingly rare; in our experience it has never happened: cases, however, have been reported in which it was necessary to plug the rhino-pharynx. If the tonsils are hypertrophied and demand removal, this is done by us after the adenoids have been operated on; except in cases in which the tonsils are enormous and impede the administration of the anaesthetic or the removal of the adenoids, in which case they should be removed before the adenoids.

For tough growths we find it necessary in very rare cases to use cutting forceps, such as Loewenberg's. With the left forefinger in the rhino-pharynx the forceps are guided to the portions of growth to be removed, care being taken not to include mucous membrane or any of the normal structures.

The after-treatment is very simple; the patient is kept in bed for twenty-four hours, and fed on cold bland food, such as milk, custard, pudding, beaten-up eggs, and jelly. The temperature is sometimes slightly febrile the first night, and the throat rather sore; but this is very transitory and slight, and is relieved by sucking ice. The bowels should be well moved. For the next two days the patient is confined to his bedroom, and for two days more to the house. No cleansing of the parts is necessary or advisable. A nasal or post-nasal douche should never be employed, as there is great risk of setting up otitis media by their use. Since giving up cleansing of the parts, and all after-treatment of the rhino-pharynx, we have hardly ever seen otitis media. For the first two or three days, owing to the irritation and inflammation set up by the operation, the nasal obstruction and deafness may be but little improved.

As regards the ears, if the membrane be simply depressed, it may suffice to inflate the middle ear by means of Politzer's bag; or, that failing, by the Eustachian catheter, for a variable period. This should not be undertaken till nearly a week after the removal of the growths, lest any blood, mucopus, or disintegrating tissue be driven into the Eustachian tubes. But with middle-ear disease the prognosis must be guarded, especially if perforation of the drum-head and chronic otorrhoea be present—serious complications which require their appropriate treatment. The nasal catarrh usually subsides in the course of a week or ten days.

If, owing to long-engendered habit, the child should continue to breathe through the mouth even after operation, this may be combated by the use of Guye's "contra-respirator" or methodical breathing exercises, which are very advantageous, when the obstruction has been actually removed; but that these exercises can be regarded as a substitute for operation, when this is indicated, has been shewn by one of us (F. S.) to be a complete delusion.

Felix Semon.
P. Watson Williams.

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F. S.
P. W. W.

II.—DISEASES OF THE PHARYNX

PHARYNGOSCOPY.

DISEASES OF THE PHARYNX AND TONSILS.
- PHARYNGITIS—
  - ACUTE.
  - CHRONIC.
- CHRONIC HYPERPLASIA OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT.
- ACUTE SEPTIC INFLAMMATIONS OF THE PHARYNX AND LARYNX.
- RETROPHARYNGEAL ABSCESS.
- HAEMORRHAGE.
- HERPES AND PEMPHIGUS.
- ULCERATIVE PHARYNGITIS.
- ACUTE MEMBRANOUS ANGINA. VINCENT'S ANGINA.
- THRUSH.
- PHARYNGOMYCOSIS.
- KERATOSIS.
- ACTINOMYCOSIS.
- GLANDERS.

DISEASES OF THE PHARYNX AND TONSILS—*continued*
- TUBERCULOSIS.
- LUPUS (*vide* p. 203).
- LEPROSY (*vide* p. 205).
- SYPHILIS.
- SCLEROMA.
- GOUTY AFFECTIONS OF THE THROAT.
- RHEUMATIC AFFECTIONS OF THE THROAT.
- NEW GROWTHS.
- NEUROSES.

DISEASES OF THE UVULA.

DISEASES PECULIAR TO THE TONSILS—
- INTRODUCTORY REMARKS.
- ACUTE TONSILLITIS.
- ACUTE ULCERATIVE TONSILLITIS.
- CHRONIC ENLARGEMENT OF THE TONSILS.
- TONSILLOLITHS.
- DISEASES OF THE LINGUAL TONSIL.

II.—DISEASES OF THE PHARYNX

By Sir FELIX SEMON, K.C.V.O., M.D., F.R.C.P., and P. WATSON WILLIAMS, M.D.

Pharyngoscopy.—The pharynx and fauces may be examined by direct inspection in bright daylight, or by the aid of artificial light reflected and concentrated by a forehead mirror, which should be the same as that used in laryngoscopy. The remarks on the form of the forehead mirror and the best kind of light will be found on page 179.

In examining the pharynx, we sit facing the patient with the forehead-reflecting mirror over the right eye, so adjusted that the eye looks through the aperture in the centre. The lamp, if one be used, should be placed on the patient's left, on a level with his ear, and so that the light is directed towards the forehead mirror and thence into the patient's mouth.

The patient should then open his mouth and go on breathing quietly, when in many cases a good view of the fauces will be obtained; generally, however, it is necessary to depress the tongue with a spatula or some form of depressor, such as Türck's or Fränkel's. In introducing the depressor it should be placed just beyond the dorsum of the tongue, and then gently and steadily depressed; if it is not placed far enough back, the dorsum of the tongue bulges up and impedes the view; whilst, if it is placed too far back, retching and nausea are induced. If the tongue is forcibly arched up, gentle pressure should be continued for a moment; if we attempt to depress the organ forcibly, it will arch up the more.

In the first place, the condition of the parts during quiet respiration should be noted. The tonsils are seen lying between the anterior and posterior pillars of the fauces; they should not project beyond the faucial pillars. Behind the faucial opening the posterior wall of the pharynx comes into view. The colour and surface of this part and of the soft palate should be noted. The patient should then be instructed to sound "Ah! ah!" and the power of retraction of the velum palati observed.

A laryngeal mirror should then be introduced as in laryngoscopy; but the mirror should be held less obliquely, so as to reflect the back of the tongue and the upper surface of the epiglottis; by this means we observe the condition of the lingual tonsil. Simple enlargement and tortuosity of the superficial veins at the back of the tongue are very common, and have no clinical importance. The lower portion of the pharynx and the beginning of the oesophagus are seen by placing the mirror in the position for laryngoscopy, while the patient's tongue is protruded and held by a cloth in the examiner's left hand. The lowest region of the pharynx, namely, the laryngo-pharynx, or that portion which lies between the level of the upper border of the epiglottis and the opening of the oesophagus, can be inspected by depressing the tongue forcibly, or by the laryngoscopic mirror, but only as far as the upper border of the cricoid cartilage, unless resort be made to von Eicken's method of hypo-pharyngoscopy or to Kirstein's or Killian's tubes. These regions belong more properly to the larynx, and for the various methods of obtaining inspection of the parts reference should be made to the descriptions given on pages 299 *et seq.*

The clinical importance of the supratonsillar fossa, to which Paterson and also Mosher have drawn attention, should not be forgotten, and in cases in which the tonsils are enlarged or subject to inflammatory exacerbations this region should be carefully noted. The plica tonsillaris, or the fold of mucous membrane which is prolonged from the anterior pillar of the fauces, is lost above the edge of the velum palati and disappears below in the root of the tongue; this structure is frequently folded over the tonsil to such an extent that the outlet of the fossa is more or less closed in. In most cases the posterior boundary of this fossa is formed by the upper part of the tonsil, which is prolonged upwards in the form of a spur, whilst the anterior boundary is formed by the anterior pillar of the fauces and the upper part of the cavity extends for a variable distance into the soft palate.

Continuing Dr. Paterson's description, we must remember "that the part of the tonsil uniting the fossa shews an arrangement of structure which differs somewhat from the remainder of the gland—a point of some importance. The lymphoid tissue is disposed in a very loose open network, which contrasts with the compact structure of the lower part of the gland, where it feels firm and is closely packed together, and presents on its surface openings of small size." Its crypts tend to find an outlet in the fossa, and as a result often contain plugs which readily undergo decomposition and may give rise to a calculus or "tonsillolith." The fossa may extend downwards and outwards behind the anterior palatal arch to the lower jaw, or extend outwards on the deep or external aspect of the tonsil, or upwards into the soft palate.

Next, the back of the uvula and soft palate and rhino-pharynx should be examined with the rhinoscope, as in posterior rhinoscopy, in order to ascertain the condition of the posterior surface of the soft palate; for, particularly in syphilis, extensive infiltration and ulceration may affect its posterior surface only, without there being anything strikingly abnormal anteriorly, beyond some hyperaemia or defective mobility. In cases of nerve disease it is necessary to use a probe to test the tactile sensibility and reflex irritability of the soft palate.

Finally, it is sometimes desirable to make the patient "gag," by introducing the tongue depressor rather farther back than usual, as in this way we cause the pharyngeal muscles to contract and to bring the tonsils well into view; thus considerable hypertrophy of these structures may sometimes be revealed, or such thickening of the lateral pharyngeal walls as we find in pharyngitis lateralis and in gouty pharyngitis.

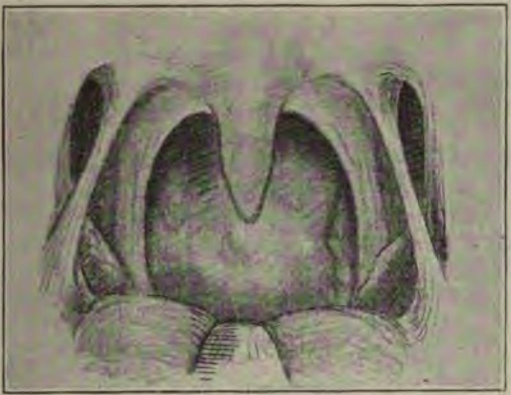

Fig. 15.—Congenital fenestration of the anterior faucial pillars. (P. W. W.)

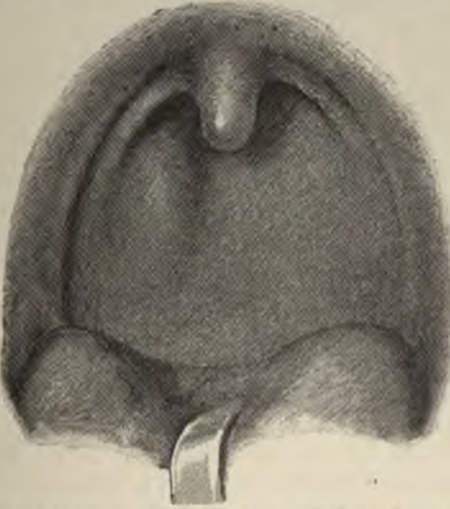

Fig. 16.—Large pulsating vessel on the posterior wall of the pharynx (Brown Kelly).

Congenital malformations are occasionally met with; the most common being a more or less completely bifid uvula, or complete absence of the uvula in association with cleft palate. The anterior pillars of the soft palate may have a separate and complete fold of mucous membrane covering the palato-glossus muscle, with either a unilateral or bilateral fenestration of the mucous membrane of the anterior pillar of the fauces which may be mistaken for perforation resulting from former disease.

Accessory thyroid glands have also been recorded, forming smaller or larger tumours in the region of the lingual tonsil.

Large pulsating vessels on the posterior wall of the pharynx occur with far greater frequency than the few reported cases would seem to imply; Dr. Brown Kelly has collected notes of over 50 cases, and, as he points out, the failure to recognise their presence may have serious consequences "if operative measures are carried out in their immediate neighbourhood." Usually these large vessels cause no abnormal sensations in the throat, and without a careful inspection they would easily escape observation. An example of such a case in a woman aged seventy-five is illustrated in Fig. 16. Dr. Brown Kelly considers that some, if not all, of the cases are due to a tortuous condition of the internal carotids, such as those shewn in Fig. 17. He has seen several instances of the condition in little children from four to six years of age. Schmidt has seen several cases, Dr. M'Bride has reported 2 cases, and many other

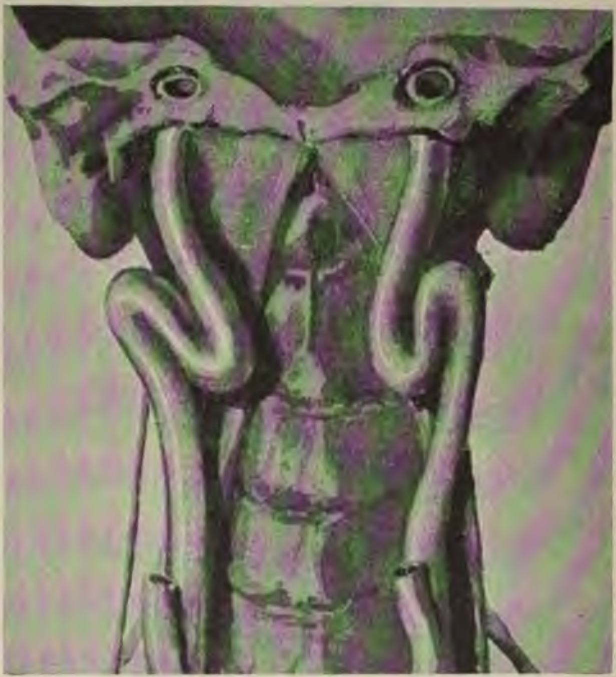

FIG. 17.—Symmetrical tortuous internal carotid arteries. Specimen in the Museum of Glasgow University (Brown Kelly).

records of single observations have been published. The pulsating vessel is not rarely the ascending pharyngeal artery.

In conclusion, we cannot too strongly insist on the importance of paying attention to the general condition of every patient who consults his medical adviser for a throat affection. A chronic pharyngitis may arise from cardiac valvular disease or diabetes, whilst gout, rheumatism, anaemia, and dyspepsia are prolific causes of acute and chronic pharyngitis; again, congestion of the pharyngeal mucous membrane or haemorrhage from rupture of small vessels may be due to gout, chronic renal disease, mitral stenosis, or portal obstruction.

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Diseases of the Pharynx and Tonsils

Acute Catarrhal Pharyngitis.—The causes may be classified as follows: (i.) *Idiopathic*, due to sudden exposure to cold and damp, especially after being in heated rooms; (ii.) *Diathetic*, especially gouty and rheumatic—many of the cases of so-called simple catarrhal pharyngitis, following exposure to damp, belong to this class; (iii.) *Toxic*, due to the action of various drugs, as, for example, antimony, mercury, belladonna; (iv.) to the virus of infectious diseases; or (v.) *Traumatic*, from burns, scalds, external violence, and the like.

Pathology.—How cold and damp may cause acute angina is uncertain; but acute catarrhal pharyngitis is frequently epidemic and often contagious, especially in the spring and autumn; this prevalence points to a microbial origin of many forms of acute catarrhal angina hitherto regarded as idiopathic and due directly to cold, and recent bacteriological researches corroborate this view. The very intimate connexion between pharyngitis, acute tonsillitis, and the rheumatic diathesis, the fact that all these affections are prone to occur under similar climatic and telluric conditions, and also that acute tonsillitis and rheumatism are probably due to infection by micro-organisms, favour the view that the so-called idiopathic and rheumatic forms of pharyngitis and rheumatic fever stand in much the same relation to one another as does the sore throat which prevails during epidemics of scarlet fever to scarlet fever itself.

Acute septic inflammations of the pharynx and larynx are due to certain varieties of infecting organisms of a virulent pathogenetic type, and all are probably, in this sense, pathologically identical. For the description of this group of cases of acute pharyngitis reference should be made to the special section on page 117.

On the other hand, there is no reason to believe that the toxic forms of acute pharyngitis are in any way associated with micro-organisms; they are more probably due to bio-chemical alterations in the tissues, similar to those resulting from the action of belladonna in acute poisoning by this drug.

Whatsoever the exciting cause of the inflammatory condition, the pathological changes in the pharynx are similar in all, and consist at first in general hyperaemia and round-celled infiltration of the affected region, with diminished secretion from the mucous glands, giving place in the course of twelve to twenty-four hours to increased secretion of greyish, viscid mucus which soon becomes muco-purulent. The implicated mucous membrane appears red, velvety, and thickened, and the uvula especially is prone to be thickened, elongated, and oedematous. In the acute septic inflammations there often is a peculiar bluish tint (*vide* Fig. 1, Plate III.). As the inflammatory condition subsides, the mucous membrane generally regains its normal colour and functions; but, on the other hand, a subacute catarrhal inflammation may persist for a considerable time, and in the absence of appropriate treatment may eventually pass into the chronic form.

The *symptoms* vary in degree according to the severity of the attack; in many cases they are slight and the patients do not seek advice. In the earlier stages a dry soreness in the throat and between the throat and nose is felt, especially during speaking or swallowing, with a sensation of stiffness in the parts, rendering speech uncomfortable. When resulting from a chill there may be some aching in the limbs and back, generally malaise, and slight fever. The dryness and harshness of the throat are due to the arrest of the secretions; after a day or two a small quantity of tenacious purulent mucus is secreted; but this is rarely so excessive in amount as in chronic pharyngitis. The tonsils and uvula are generally more or less implicated, and are red and swollen, or in the severer cases dusky purple in colour; the catarrhal inflammation often spreads up to the rhino-pharynx, and to the Eustachian tubes, giving rise to temporary deafness; or it passes downwards to the larynx and trachea.

Diagnosis.—It must be borne in mind that diphtheria, scarlet fever, measles, and septic inflammations may begin with symptoms of acute pharyngitis, and therefore all cases of acute pharyngitis, especially in children, should be watched. It is very important, from a therapeutic standpoint, to recognise the cases in which the affection is due to rheumatism and gout.

Treatment.—In milder forms very simple treatment is generally sufficient, such as a hot mustard and water foot-bath and a Dover's powder at bedtime. A menthol spray (℞ Menthol ʒss., ol. adepsin. pur. ℥j.), sprayed several times a day by means of an oil atomiser, and sucking ice will greatly relieve the local inflammation. When the larynx and trachea are affected, the inhalation of tincture of benzoin, or a mustard poultice applied to the chest are serviceable. The bowels should always be freely moved by saline aperients. For the rheumatic cases salicin compounds, and for the gouty aspirin, colchicum, and alkalis are required. As the local inflammation subsides we may prescribe the compound krameria pastille. Local astringent applications are rarely necessary.

Chronic Pharyngitis.—The causes of chronic pharyngitis are many and diverse, and often enough they are remote and obscure. Catarrhal attacks, scarlatina, or measles may leave behind them hypertrophy of the lymphoid nodules of the pharyngeal mucosa itself. At puberty chlorosis and general anaemia, dyspepsia, and constipation are fruitful causes of granular pharyngitis—the first two conditions being perhaps the most frequent; later in life dyspepsia, gout, rheumatism, diabetes, the irritation of tobacco smoke, alcoholic drinks, and so forth, operate in a like manner; but the pharyngitis in these cases is accompanied by general irritation and congestion of the whole pharyngeal mucous membrane; consequently, whilst on the one hand we may meet with enlarged lymphoid nodules only, as in granular pharyngitis, in these latter conditions there is also general thickening of the mucosa, with enlargement of the vessels and secretion of tenacious mucus in the rhino-pharynx and pharynx.

If the patient suffered in childhood from post-nasal adenoids which have not completely atrophied, and chronic nasal catarrh has persisted, there is often a copious collection of unhealthy sticky mucus in the rhino-pharynx, and the condition is known as post-nasal catarrh. In some cases the disease is congenital in origin. Whilst the characteristic objective conditions in the pharynx are sometimes seen in very young children, the subjective symptoms do not usually arise before the age of eighteen onwards. In children the lymphoid tissues are especially active; and not only are the palatine and rhino-pharyngeal tonsils well developed, but the same excess is found, though in a lesser degree, in the smaller aggregations of lymphoid tissue around the muciparous glands. Thus, it is impossible to separate completely the simple catarrhal and chronic hypertrophic forms; they generally coexist, though the characteristics of the one or the other may predominate.

Some cases, especially those due to constipation and dyspepsia, or to portal congestion, may be regarded as toxic in origin, and due to a failure on the part of the liver to arrest and destroy toxins resulting from imperfect digestion or decomposition in the intestinal tract; these toxins, like belladonna, have a specific effect on the pharyngeal mucous membrane (this opinion has been held for some length of time by one of us—W. W.). The soreness, stiffness, and hyperaemia, the dryness of the throat and pain in deglutition, which are characteristic of belladonna or muscarine poisoning, are simulated very closely by the sore throat of dyspepsia following a late and heavy meal; and these conditions, by frequent recurrence, even in a mild degree, eventually bring about permanent structural alterations of the mucous membrane.

We may explain the occurrence of gouty pharyngitis in much the same way.

The pain, so often complained of, is generally attributed to the implication of the nerve-endings in the degenerated granules; it is, however, more probable that the nerve filaments are irritated by the same causes which produce such very obvious hyperaemia and thickening of the mucous membrane; but the factor of temperament is clearly seen in the painful character of the chronic pharyngitis in chlorotic girls and in those of the neurotic temperament.

In later life the pharyngeal mucous membrane may become more or less atrophied, and the secretion of mucus very deficient—a condition sometimes distinguished by the term *atrophic pharyngitis* or *pharyngitis sicca*. This *dry* form is a not infrequent complication in diabetes mellitus.

Thus it will be seen that chronic pharyngitis is generally due to several factors acting conjointly, which may be classified as follows:—

(i.) One of the most important is general anaemia. Granular pharyngitis is most frequently met with in anaemic girls, in whom also other signs of chlorosis exist.

(ii.) The "strumous," rheumatic, and gouty diatheses. Gouty pharyngitis is usually characterised either by general or, more frequently, by lateral thickening, which often gives the appearance of thickened bands of tissue extending down the lateral walls of the pharynx behind the posterior palatine pillars.

(iii.) Dyspepsia, especially if associated with constipation or portal congestion, whether due to gastro-intestinal catarrh or heart disease, and constipation are prolific causes.

(iv.) Diabetes.

(v.) Constant exposure to dust or irritating vapours, as in mattress-making, stone-dressing, tobacco-manufacturing.

(vi.) Abuse of alcoholic drinks and of tobacco-smoking; excessive use of irritating condiments.

(vii.) Recurrent acute attacks of catarrhal pharyngitis, or measles, scarlet fever, and other exanthems.

(viii.) Improper methods of voice production, resulting in congestion of the mucous membrane of the fauces; and excessive use of the voice during an attack of acute or subacute pharyngitis.

Symptoms.—In making a diagnosis of chronic pharyngitis, it is very important to remember that every departure from the ideal normal pharynx does not constitute disease; that in fact nearly all the objective conditions observed in this affection may be present without producing symptoms, and that in this disease there is no constant relation between the physical signs and the subjective symptoms. The patients are apt to complain of a constant irritating cough, and a sensation as of a hair or foreign body in the throat which they cannot get rid of; or of soreness and aching often amounting to sharp pain, especially in swallowing: often also there is a sense of weakness and discomfort in the fauces. The symptom for which advice is most usually sought, however, is impairment of vocal power. Hence professional and amateur singers, clergymen, public speakers, lawyers, and schoolmasters form by far the largest contingent of those who seek advice on account of chronic pharyngitis. Their complaints are various. Most frequently it is stated that the voice is readily tired and deficient in resonance and timbre; singers usually complain of deficiency or even of loss of the higher notes. These alterations in the voice are even more marked in those younger patients in whom post-nasal growths occupy the rhino-pharynx. Prolonged speaking, or singing, in the presence of marked chronic pharyngitis, often results in aching in the throat and back of the neck, whilst the voice gets weaker and weaker. After this has continued for a time the larynx becomes more or less congested, and then the voice, for public speaking, often goes altogether.

In patients who are suffering from simple catarrhal pharyngitis the chief features are the constant accumulation of mucus in the throat, the necessity for perpetual hawking, and the tendency to gagging and retching.

On examining the pharynx the mucous membrane is found to be diffusely congested. In the simple catarrhal forms it is pink, with enlarged venules coursing over the posterior wall, which is often more or less covered by collections of mucus. The mucous membrane of the uvula and soft palate is sometimes considerably congested, thickened and granular, and some enlarged mucous glands are seen. The tonsils are often somewhat enlarged, with gaping crypts, some hypertrophied lymph-follicles are almost always observable; in granular pharyngitis there is often little else to be seen. There is seldom any excessive accumulation of mucus; in fact the complaint very often is that the throat is too dry. In some cases, and particularly in the gouty, two longitudinal lateral bands of hypertrophic tissue stand out prominently; this is important from the diagnostic point of view. Not rarely the larynx participates in the process, in the shape of congestion, relaxation, and granular appearance of the vocal cords, with corresponding huskiness and fatigue on exertion.

Treatment.—Before entering on the question of treatment it is desirable to lay stress on the necessity for attending to any primary dyscrasia, instead of relying solely on local treatment. Thus, in the great majority of cases, the general treatment of chronic pharyngitis is of far greater importance than the local. Anaemia and chlorosis must be combated with Blaud's pills and aperients; gout and rheumatism, diabetes, constipation, dyspepsia, and portal congestion should each receive its appropriate treatment; whilst in other cases, as in many clergymen and schoolmasters suffering from granular pharyngitis, the health is more or less impaired and general nervine tonics are indicated, though, as a rule, the relief is unfortunately only temporary. Many patients will be greatly benefited by a course of alkaline or aperient waters, such as those of Aix-les-Bains, Ems, Mont Dore, La Bourboule; and, for gouty patients, Bath, Strathpeffer, Harrogate, Kissingen, Marienbad, or a gentle Carlsbad course is advisable, particularly in the case of women suffering from chronic pharyngitis during the climacteric period.

Local treatment, however, is often required. The usual astringent lozenges, sprays, pigments, and gargles are, as a rule, most disappointing and inefficient. A very useful spray for general use in relaxed throat is a pinch of salt dissolved in a wineglassful of cold water. If the mucus tend to collect in the pharynx and rhino-pharynx, a solvent coarse spray, composed of bicarbonate of sodium (1 to 2 per cent) with a few grains of boracic acid, may be used once or twice daily. A pastille containing 2 grs. of guaiac resin, $\frac{1}{2}$ gr. powdered cubebs, $\frac{1}{20}$ gr. of emetine, and $\frac{1}{4}$ gr. of menthol, slowly dissolved in the mouth four or five times daily, will often relieve rheumatic forms of pharyngitis, whilst $\frac{1}{4}$ of a grain of codeine in the form of a pastille, and repeated if necessary, is useful in relieving the constant cough which in some cases of irritable pharyngitis interferes with sleep.

Enlarged granular lymphoid follicles should be destroyed by the galvano-cautery. Having previously cocainised the part (with a 20 per cent solution of alypin or cocaine), a flat platinum or porcelain burner is placed on the centre of a granule when cold; the current is then turned on to a cherry-red heat and immediately withdrawn. If there are any enlarged veins on the posterior pharyngeal wall they may be divided in places in a similar manner, so as to obliterate them; if left, they tend to maintain the vascular engorgement and general congestion. After using the galvano-cautery the patient should only take bland or cold food for a day; sucking ice may be grateful to him for a few hours after operation, or an insufflation of anaesthesine or orthoform may be used to relieve pain and soreness. Six to ten spots may safely be cauterised at one sitting. A pellicle forms on each cauterised spot, which separates in a day or two, leaving a clean surface. The cauterisation may be resumed after an interval of three days to a week, till all the granules have been destroyed in turn.

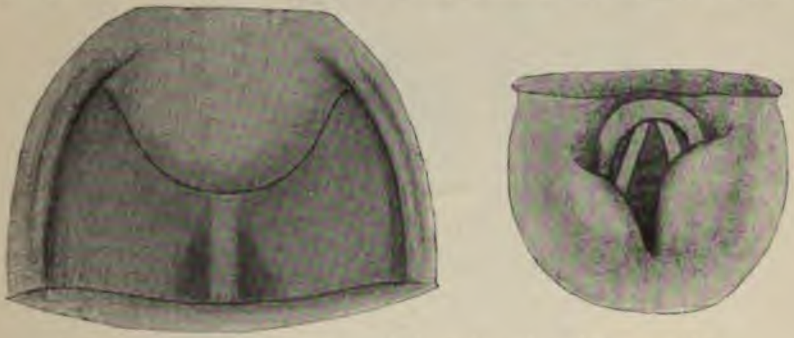
Thickened bands of mucous membrane, when present, should likewise be destroyed by the galvano-cautery. Other methods of destroying the granules can only be recommended when the galvano-cautery is not available. The best alternative is to touch the centre of each with chromic acid fused on a silver probe.

Great stress must be laid, of course, upon the future avoidance of those causes of irritation to which the malady was due, such as improper use of the voice, insufficient exercise, dietetic errors particularly in those who are gouty, and, above all, abuse of alcohol or excessive smoking.

Chronic Hyperplasia of the Mucous Membrane of the upper Respiratory Tract.—A peculiar form of chronic thickening of the mucosa is sometimes encountered, in the pharynx especially, but also in the nasal passages and larynx. Dr. Brown Kelly, who first recorded a case of this affection under the name sclerotic hyperplasia, considered from the histological evidence that it was analogous to simple subglottic hyperplastic laryngitis, and that it was distinct from the very similar subglottic hypertrophy that is common in scleroma and is occasionally seen in syphilitic cases. One of us (F. S.) has described three cases of this chronic hyperplasia, and does not consider that the condition is of a sclerotic nature, as it is capable of spontaneous retrogression.

Morbid Anatomy.—The disease is characterised by a uniform thickening of the affected areas, which present a peculiar yellowish, lardaceous appearance, the infiltration being smooth and semi-solid. The uvula becomes enormously thickened, and the soft palate thick and hanging like a curtain, whilst the infiltration of the faucial pillars or portions of the submucosa of the pharynx forms thick bands extending down to the oesophagus, and interfering more or less with deglutition.

When the larynx is affected the thickening sometimes invades an arytaenoid region, in other cases the epiglottis and aryepiglottic fold. The aperture of the naso-pharynx was reduced in Dr. Brown Kelly's case so as barely to admit a finger, and like the bands of the pharynx the mucosa was smooth, grey, and of the consistency of muscle. There is a tendency to improvement and lessening or disappearance of the infiltration in the course of years, whilst the progress of the disease is unaffected by antisyphilitic treatment.



Figs. 18 and 19.—Dr. Brown Kelly's case of sclerotic hyperplasia of the mucosa of the pharynx and larynx.

The *etiology* is obscure; there is no evidence of infection by specific micro-organisms, and tuberculosis, syphilis, and scleroma seem to be quite out of the question in them all.

We are enabled to reproduce Dr. Brown Kelly's illustration of the first recorded case, and we quote from Mr. Shattock's pathological report of pieces of the affected uvula in one of Semon's cases: "The uvula is generally enlarged, its cross-section having a diameter of 9 mm., and its length being correspondingly increased. The investing epithelium is intact and normal; between its elements a few polymorphonuclear leucocytes have wandered from the subjacent tissue. The increase in size is due to a diffuse formation of finely fibrillar connective tissue, in the centre of which there occur small groups of fat-cells. The proper corpuscles of the new connective tissue have the usual characters and are moderate in number. The uniformity of the microscopic picture is only broken by denser collections of cells which bear an obvious relation to the smaller blood-vessels, and are almost confined to the periphery of the cross-section, though here and there similar collections occur in the more central parts. The cells composing the clusters consist of lymphocytes and intermingled plasma-cells (Unna). Neither mast-cells nor eosinophil leucocytes are present in the section. Unna's acid orcein reveals a mesh of fine elastic fibrils pervading the connective tissue. The arterioles are everywhere normal. At the periphery of the sections, a short way beneath the epithelium, a certain number of the capillaries which lie in the small-celled infiltration are plugged as a result of endothelial proliferation. No micro-organisms are demonstrated by Gram's or other methods. This is equally the case when the decolorising action of alcohol is evaded after staining with carbol fuchsin, by the use of glycerin as a mounting medium. The examination shews no more than the anatomical character of the condition, it does not demonstrate its cause. The lesion presents none of the structural features of tuberculosis, syphilis, or rhinoscleroma. The overgrowth of connective tissue has no relationship with a neuro-fibromatous pachydermia, since the nerves are not involved in the general fibromatosis.

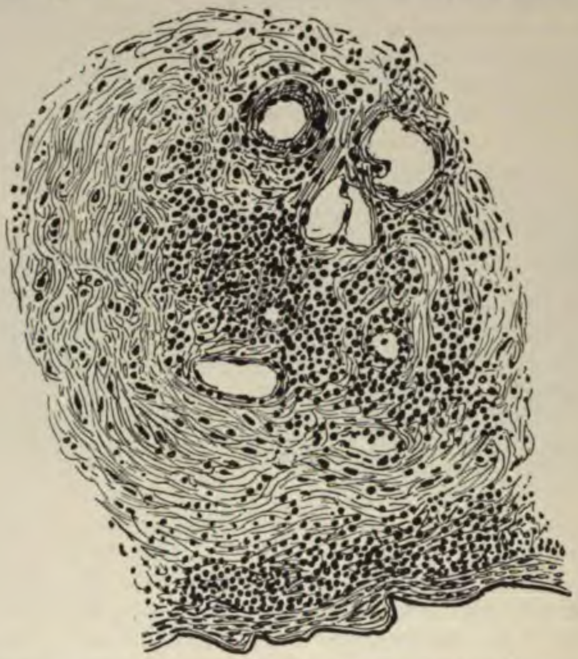


FIG. 20.—Low power section of the uvula of Dr. Brown Kelly's case, which almost entirely consisted of dense fibrillated connective tissue of sparsely cellular character.

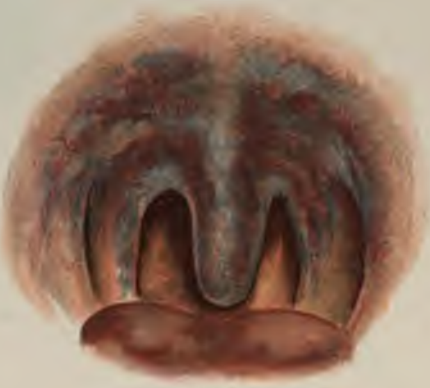


Fig. 1.

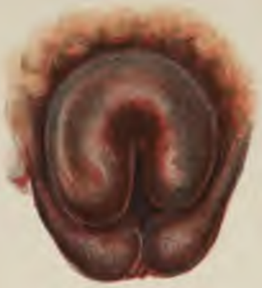


Fig. 2.

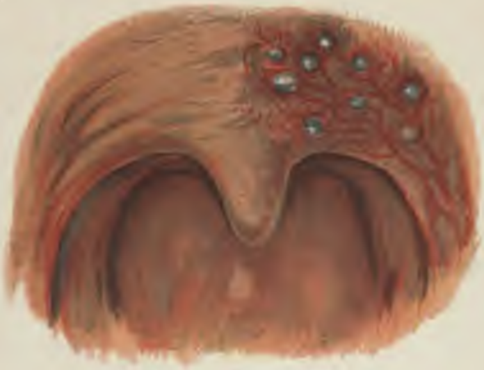


Fig. 3.

PLATE III.

ACUTE SEPTIC PHARYNGITIS AND LARYNGITIS, AND HERPES OF THE SOFT PALATE.

Fig. 1. Acute Septic Pharyngitis. The bluish tint of the oedematous inflamed tissues should be noted.

,, 2. The same in the larynx.

,, 3. Herpes of the soft palate.

The enlargement, again, is not due to a dilatation of lymph spaces, as in a lymphangiomatous or lymphangiectatic lesion. Nor does it bear relegating to the group of angioneurotic oedemas observed in the skin, since the lesion was not sudden, either in its onset or at any time in its rate of extension. The complete absence of chronic arterial change further removes any relationship between the disease and erythromelalgia, seeing that in the cases of the last-named condition which have been histologically examined, local arterial disease has been observed. The histological changes approach most nearly to those met with in hyperplastic rhinitis in its later, or what has been called its secondary, stages; in this are encountered the same overgrowth of firm fibrillar connective tissue and similar small-celled infiltration around the lesser vessels."

REFERENCES

1. Kelly, Brown. "Sclerotic Hyperplasia of the Pharynx and Naso-Pharynx," *Lancet*, 1901, i. 995.—2. Semon, F. "Chronic Hyperplasia of the Mucous Membranes of the upper Respiratory Tract," *ibid.*, 1905, i. 484.

Acute Septic Inflammations of the Pharynx and Larynx (including Phlegmon of the Cellular Tissue of the Neck—Angina Ludovici).—Under this heading we include a number of forms of acute septic inflammations of the pharynx and larynx which formerly were usually considered as pathologically different, such as acute inflammatory oedema of the pharynx and larynx, phlegmon of the pharynx and larynx, and erysipelas of these parts. In our opinion phlegmonous cellulitis of the neck (angina Ludovici) also comes under this head. The word "septic" may possibly be objected to as rather vague in its meaning, but to substitute for it "infective" would lead to confusion, as it would then include the changes due to the organisms of tuberculosis, diphtheria, and syphilis, which are not concerned in the process here described; "pyogenetic" would be equally inadmissible, as the inflammation is not necessarily suppurative. The fact of the matter is, that, as became evident in the discussion at the Royal Medical and Chirurgical Society, when the subject was first brought before it by one of us (F. S.) in 1895, a really satisfactory terminology does not exist as yet with regard to this class of cases.

In a communication to the Royal Medical and Chirurgical Society,[1] one of us (F. S.), in 1895, argued, on clinical and bacteriological evidence, that these various forms of acute septic inflammation of the throat should be considered as varying degrees of virulence of one and the same pathological process. The primary seat and subsequent development depend in all probability upon accidental breaches of the protecting surface through which the pathogenetic micro-organism, which causes the subsequent events, finds an entrance; and it is absolutely impossible to draw any definite line of demarcation between the purely local and the more complicated cases, or between the oedematous and the suppurative forms. That each and all of these septic processes may be produced by several pathogenetic organisms does not, in our opinion, in the least militate against their pathological identity. These micro-organisms are "interchangeable" in the sense that each and all of them, when penetrating into the tissues, produce one and the same effect, namely, an acute septic inflammation—oedematous, purulent, or gangrenous. Likewise we believe that erysipelas etiologically considered, is not a specific disease; usually it is caused by the *Streptococcus pyogenes*, but it may also be produced by the *Staphylococcus pyogenes aureus*, as Max Jordan's researches have definitely proved. The micro-organisms causing erysipelas most probably enter into the circulation in every case; pyaemia following erysipelas is therefore primary, and not due to a mixed infection.

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[1] We must refer readers interested in this subject to this paper (*Med.-Chir. Trans.*, London, 1895, lxxviii. 181) and to its discussion (*Proc. Roy. Med.-Chir. Soc.*, 3rd ser., vol. vii. p. 134) for the particulars which considerations of space will not allow us to discuss at length in this article.

When we attempt to draw definite distinctions between the inflammations associated with different micro-organisms we fall inevitably into a confusion of terms. In the discussion on Semon's paper the late Prof. Kanthack gave most valuable support to our views by quoting in detail four cases of his own in which various pyogenetic micro-organisms had been found producing various stages of the same process. Thus these cases bacteriologically distinct were pathologically identical (see also Vol. I. pp. 533, 536, 1st edit., 1896). Since then Mr. de Santi has brought forward valuable evidence, afforded by four cases, supporting these opinions. One of these cases refused treatment, but brief notes of the remaining three will be instructive: (*a*) Case of Bezold's mastoiditis, an operation on the mastoid being followed by cutaneous erysipelas and acute septic inflammation of the faucial pillars, the soft palate, and uvula. Cultures from the throat shewed mainly *Streptococcus pyogenes*, with some staphylococci. On each of three successive days 10 c.c. of antistreptococcic serum were injected, and the temperature fell from 103·4° to 99·2° F. By the seventh day the throat was normal. (*b*) Case of acute gangrenous inflammation of the throat, the uvula being enormously swollen, intensely red, and the lower half black and gangrenous. The soft palate and tonsils and pharynx were also greatly inflamed, and in the larynx the left aryepiglottic fold was enormously swollen and oedematous, the epiglottis being implicated in less degree. Cultures revealed the presence of *Streptococcus pyogenes* and some staphylococci. Five hours after injection of 20 c.c. antistreptococcic serum, the patient was able to swallow. The next day there was marked improvement locally and generally, and he made an uninterrupted recovery without any further injection. (*c*) Acute pharyngitis due to *Streptococcus pyogenes*, followed by septicaemia and pericarditis. Within the next forty-eight hours four injections of 10 c.c. of serum each were given; the effect was remarkable, the patient beginning to rally at once.

Etiology.—The infections here discussed are due to the invasion of the system by pathogenetic organisms, of which, so far, the *Streptococcus pyogenes* appears to be the most frequent. No doubt, however, any one of the other pyogenetic microbes, such as *Staphylococcus aureus* or *citreus*, *Pneumococcus*, *Micrococcus tenuis*, *Bacillus coli communis*, the *Bacillus pyocyaneus*, if by chance it multiply sufficiently, may alone produce an acute septic inflammation indistinguishable, except from a bacteriological point of view, from the streptococcic inflammation.

Morbid Anatomy.—Pathologically these inflammations are characterised by a violent exudation into the tissues affected. This exudation may be serous, sero-purulent, purulent, and in the worst cases may even lead to gangrene. All these various forms, however, merely represent various degrees of intensity of inflammation, not differences in kind.

Symptoms.—For clinical purposes we may recognise four degrees of inflammation: (*a*) Superficial septic inflammation, as in the so-called "hospital sore throat"; (*b*) oedematous inflammation, as acute oedematous tonsillitis, uvulitis, pharyngitis, epiglottiditis, arytaenoiditis, cellulitis of the tissues of the neck, and so forth; (*c*) suppurative inflammation or phlegmon; (*d*) gangrenous inflammation.

Septic inflammations of the throat attack persons of all ages and both sexes, very frequently even those apparently in perfect health; though in those who are run down in health from any cause, or are suffering from some debilitating affection such as diabetes, the disease is especially prone to occur and to run a severe course.

We know nothing definitely about the length or even the existence of an incubation stage. Prodromal symptoms, such as headache, feverishness, sore throat, and general malaise, may precede the onset of more acute symptoms for a few days. In the slighter forms, as in hospital sore throat, there may only be localised soreness and stiffness in the throat, with headache and general malaise, without fever or marked constitutional disturbance. These mild cases, however, may pass into the more severe forms. In the grave forms often enough the disease manifests itself quite abruptly. It may be ushered in by a rigor and rapid rise of temperature. The course of the fever is very variable, as it probably depends on the virulence of the septic infection in the individual case; and, though usually ranging high, it may never rise above 100° F., especially in asthenic cases; or it may present a remittent or relapsing type: but the temperature as a rule reaches its highest point at the very onset. Rigors occurring later in the course of the disease generally indicate further complications or the onset of suppuration. The urine is febrile; the frequency of albuminuria has yet to be determined; sugar is found comparatively often. The pulse during the acute stage is usually frequent, full, and bounding, but soon becomes weak and compressible. When suppuration has occurred and the strength is greatly reduced, the pulse is small and thready, and perspiration profuse.

In those rare cases in which the nervous centres are affected early, the pulse and respiration become irregular, and the patients are generally delirious by the second or third day.

Whether the part attacked be the fauces, pharynx, larynx, or cellular tissue of the neck, the first symptom usually complained of is sudden pain in the throat and difficulty in swallowing, which within a few hours may amount to complete aphagia. If the larynx be involved, hoarseness of voice, and, soon after, laryngeal stridor and dyspnoea are observed. Often the aphonia is complete, whilst the difficulty of breathing may become extreme within a few hours from the onset of the disease. The aphagia and dyspnoea last for a few hours to a few days; but in the cases of recovery, these and all other symptoms rapidly subside.

Objectively the symptoms vary, of course, with the seat of the inflammatory process. In the great majority of cases the pharynx is first affected, and more especially the tonsils—the latter, with their anatomical configuration, forming a natural portal for the entry of infecting micro-organisms into the body. This process was fully considered in F. Semon's paper, to which we have referred. On the other hand, the microbes may pass on farther to find a point of invasion in the tissues lower down, in the larynx, especially in the epiglottis, or in the cellular tissue of the neck.

When the tonsils are primarily affected, the inflammation—clinically speaking—hardly differs from ordinary acute follicular tonsillitis. In the case of the pharynx rapid oedematous swelling occurs, and the uvula may be greatly elongated and thickened to the size of the little finger. The swelling is often distinguished by a peculiar bluish discoloration (Fig. 1, Plate III.). After a few days, if suppuration do not occur, the swelling subsides, leaving the mucous membrane more or less wrinkled in appearance; or the inflammation may spread down to the larynx. Absence of severe local symptoms may obscure the critical nature of the affection. One of us (P. W. W.) lost a medical colleague who had what appeared to be simple tonsillitis with septic epiglottiditis. A bad prognosis was given, the laryngitis increased, and in two days the patient sank from heart failure.

Some of the worst and most fatal cases begin in the pharynx and suppurate in the course of a few days, the septic inflammation remaining limited to that part (Senator's acute infectious phlegmon of the pharynx). More frequently it extends to the regions around, or spreads downwards, much more rarely upwards, to the naso-pharynx, the nasal passages, and even to the membranes of the brain. In the great majority of cases of septic pharyngitis the inflammatory process leaves this part in a few hours or days and extends downwards to the larynx. Here it appears that the epiglottis is generally most markedly affected, becoming enormously swollen and turban-shaped, so that by simply depressing the tongue it may often be seen as a semi-transparent scarlet or bluish-red roll. Next in point of frequency the arytaenoids and the arytaeno-epiglottidean folds suffer, and lose their characteristic shape in the enormous red or purple swelling which takes place; a swelling very often so great as completely to hide the ventricular bands and vocal cords (Fig. 2, Plate III.). In such cases, as already mentioned, the voice at first is weak and hoarse; in a day or two, or even in a few hours, complete aphonia and dyspnoea supervene, and the glottic chink is often so narrowed that at any moment the immediate performance of tracheotomy may become necessary. In other cases the submaxillary or cervical cellular tissues become primarily infected, the pathogenetic micro-organisms gaining entrance from the mouth by a carious tooth or fissure in the mucous membrane, by the tonsil or pharynx, and causing a hard swelling under the tongue and a localised hard, brawny infiltration beneath the jaw—hitherto commonly named angina Ludovici—but in its eventual course spreading to the pharynx or larynx, or to other regions of the neck, and ending in resolution, or more usually in suppuration; whilst in the worst cases gangrene may ensue. In some cases diffuse purulent infiltration is met with, or abscesses arise in the oedematous cellular tissue or between the muscles of the neck. In the very worst cases metastatic abscesses occur either in superficial parts or in joints. Except in its primary seat the affection is in onset, course, and event precisely similar to the disease as seen in the pharynx or larynx.

Whilst the purulent variety of the septic inflammation usually leads to speedy death, cases of serous inflammation of the larynx and its neighbourhood may get well within a few days, however considerable the inflammation. Here, again, it is characteristic that the maximum inflammation is usually attained within a few hours from its very onset; and that in the cases in which recovery takes place even considerable diminution of the swelling is the rule within a day or two from the beginning.

Often, however, the disease is not confined to the neck, but spreads, sometimes with incredible rapidity, to other parts. In addition to the lungs, in which patchy or general pneumonia may appear, the serous membranes are particularly liable to suffer; and pleurisy (single or double), pericarditis, peritonitis, or meningitis may appear within a few days or even hours from the initial rigor. As in the original seat of the disease, the exudation in the serous membranes may be either of a serous or of a purulent character; sometimes it is mixed, *i.e.* in parts serous, in others purulent; sometimes it is fibrinous. Even in cases complicated with pneumonia, pericarditis, and pleurisy, recovery is possible; and if it does occur, is remarkable for its quickness and completeness. Usually, however, in more severe cases death ensues with signs of increasing coma and heart failure; and in the worst of them the whole process from beginning to end may not occupy more than ten to twelve hours.

In very rare cases it appears as if the whole brunt of the septic infection, apart from the areas first attacked, fell upon the central nervous system. In such cases epileptiform convulsions, delirium, irregularity of the heart and pulse are amongst the earliest symptoms; and death may occur with signs of severe septic infection of the nervous system, without any thoracic complications, and after the local inflammation of the pharynx and larynx has completely subsided.

The above description represents what we at present know of the influence of these pathogenetic micro-organisms, and of the symptoms caused by them. In the belief of one of us (F. S.), however, we are only on the threshold of our knowledge of these septic affections, and some recent experiences incline him to the belief that sometimes they may, in addition to those described, cause a very different chain of symptoms and appearances, which it would be premature as yet to describe under this heading.

Treatment.—The necessity for prompt and energetic treatment in all forms of septic inflammation is but too obvious. Our aim must be directed towards combating the infection, controlling the local inflammation, supporting the patient with light nourishment, and watching for any symptoms of heart failure. For the first-named purpose it will, of course, be necessary to ascertain as quickly as possible the nature of the specific micro-organism causing the disease in a given case, in order to inject the corresponding antitoxin. Unfortunately, this requires time, and, further, we do not yet possess specific antidotes for every species of the pathogenetic micro-organisms which may cause these septic inflammations. Should circumstances permit, it will naturally be our duty first to try to determine the specific micro-organism with which we have to deal in the case under observation, and then to inject the corresponding antitoxin (if such should have already been prepared). Should this, however, be impossible, or should the disease have already lasted more than twenty-four hours before the patient comes under observation, we should not hesitate to inject at once 20 c.c. of a polyvalent antistreptococcic serum, to be followed, if necessary, by repeated injections, guided by the experience—(1) that the disease is caused more frequently by streptococcus invasion than by any other species of pathogenetic micro-organisms, and (2) that success is more probable when the antitoxic treatment is commenced at an early stage of the disease. A few severe cases appear to have been saved by the early adoption of this method; on the whole, however, it must be confessed that we are still far from being successful in the etiological treatment of these affections. As local treatment, ice may be administered internally and also externally by means of Leiter's tubes, or the ice-bag applied to the front of the neck, but whilst such applications are often comforting to the patient, and may in some measure reduce the local inflammatory swelling, cold is apt to increase the liability of the invaded tissues to necrose, and it is therefore necessary to employ these cold applications with caution and judgment. If there be oedema of the larynx, careful watch must be kept lest at any moment intubation or tracheotomy become necessary; and when dyspnoea has appeared the patient must not be left alone for a minute; in fact, dyspnoea, if at all marked, is an indication for immediate intubation or tracheotomy, unless the laryngeal obstruction can be relieved by freely scarifying the parts affected.

Four or five grains of quinine every four hours should be ordered; and if the pulse be weak, and there be any indication of heart failure, this may be combined with the tincture of perchloride of iron and digitalis. In such cases, or where pneumonia has supervened, one of us (F. S.) has found frequent inhalation of oxygen very useful. Light nourishing food must be given, and alcohol in the form of frequently repeated small doses of brandy. In suppuration, particularly in cases of phlegmonous cellulitis of the neck, the affected tissues should be incised, and the resulting wound treated antiseptically.

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Retropharyngeal Abscess.—*Causes.*—Retropharyngeal abscess is a—usually circumscribed—suppuration occurring in the tissues between the mucous membrane of the posterior wall of the pharynx and the spine. It is mainly a disease of early childhood, though occasionally it occurs in adults. The vast majority of cases must be called idiopathic, and due to inflammation of the lymphoid tissue of the pharynx, arising from no definitely assignable cause, in young children up to the age of four. The "strumous" diathesis and rickets dispose to its occurrence; or it may follow measles, scarlet fever, or injury. It is sometimes due to caries of cervical vertebrae, or, in rare cases, to burrowing of pus from other regions; it is probable that not a few cases are septic in origin, especially in older patients. The affection may follow injury from blows or foreign bodies. One of us (F. S.) has twice seen it occur in association with adenoid vegetations.

Pathology.—In children there is an aggregation of lymphoid tissue in the posterior wall of the pharynx opposite the second and third cervical vertebrae; and the suppuration is usually due to inflammation and breaking down in this tissue on one or other side: the abscess is rarely median. In adult patients the suppuration occurs in the cellular tissue which remains after the involution of the lymphatic tissues of the pharynx. The abscess is generally confined to the oro-pharyngeal region, and, though it may burrow down towards the oesophagus, it very rarely extends upwards much above the level of the soft palate.

The glands below and behind the angle of the lower jaw on the side of the abscess usually become enlarged, indurated, and inflamed.

The abscess may rupture spontaneously into the pharynx, or burrow in various directions; the inflammation very often extending to the larynx with resulting acute laryngitis or oedema.

Symptoms.—The onset may be acute or chronic. If acute, there is general pyrexia, sometimes preceded by a rigor with local heat and painful tumefaction, which, on inspection or digital exploration, appears as a fluctuating bulging of the posterior pharyngeal wall. As a rule, the voice is husky or aphonic; and cough resembling croup is usually present, accompanied by more or less acute dyspnoea and dysphagia. The child's cry has a peculiar throaty tone. Fixation of the head is generally a marked feature. In the more chronic cases the symptoms are much the same, but the temperature is not raised. In adults difficulty and pain in deglutition are the chief subjective symptoms. In children it is more difficult to detect the bulging abscess.

Diagnosis.—The symptoms in young children are easily mistaken for croupous laryngitis; but in retropharyngeal abscess deglutition as well as respiration is difficult: moreover, the fixation of the head and the unilateral swelling below the jaw point to retropharyngeal abscess. The chronic form has to be distinguished from sarcoma, which grows rapidly, does not fluctuate, often has an irregular or nodular surface, and is rarely attended by actual rigidity of the head.

The prognosis in very young children should be guarded, especially when the symptoms of laryngitis are decided; in older children and in adults the prognosis, under appropriate treatment, is always favourable. Untreated, the rupture of the abscess into the pharynx is liable to cause suffocation from the pus entering the larynx; whilst the danger of oedema of the larynx causing acute asphyxia is very considerable. It is obvious that any underlying affection, such as caries of the cervical vertebrae, would greatly modify the prognosis as regards recovery.

In the acute cases of adults there is even more need for a cautious prognosis, as they sometimes take the peculiarly fatal course and character of acute septic pharyngitis—the so-called acute infectious phlegmon of Senator.

Treatment.—In all acute cases young children should be placed in a steam tent, whilst adults should frequently use medicated steam inhalations. The treatment consists in evacuating the pus as soon as fluctuation is detected, either through the mouth by the knife, or, especially, if the case is complicated by cervical caries, by an incision behind the sterno-mastoid muscle under strict antiseptic precautions. The operation through the mouth should always be done with the patient's head hanging low down, to avoid the danger of pus escaping into the larynx. Of course, if the pus be actually pointing behind the sterno-mastoid, or elsewhere, this will determine the seat of evacuation. Aspiration is often recommended, but refilling of the abscess cavity is more likely to occur.

The great danger lies in the occurrence of oedema of the glottis. Ice should be sucked if the patient be old enough, and hot applications made to the neck and submaxillary region. Young children should be kept in the steam tent, and any symptoms of obstructive dyspnoea carefully watched for; as intubation or tracheotomy may at any moment be urgently required, even for some little time after the evacuation of the abscess.

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Haemorrhage from the Pharynx.—Haemorrhage from the pharynx is deserving of special note, not so much on account of the actual causes of bleeding in this region, but of the frequency with which patients complain of "bleeding from the throat," and of the gravity of the pulmonary disease which is only too often the real source of the loss of blood attributed to the throat.

Causes.—The chief causes of bleeding from the mouth and throat are—(*a*) Alterations in the condition of the blood in various pathological states, such as purpura, pernicious anaemia, leukaemia, mercurial stomatitis, haemophilia, renal affections, influenza, and various acute fevers, especially typhoid fever and yellow fever. (*b*) Suppuration and ulceration, as in malignant disease, lupus, or syphilis. (*c*) The oozing of blood from spongy gums or from the relatively rare condition of multiple telangiectases. (*d*) Post-nasal adenoids. (*e*) So-called vicarious haemorrhage in women at the menstrual period. (*f*) Rupture of enlarged veins in the pharynx, especially in gout, and portal cirrhosis of the liver. (*g*) Laryngeal haemorrhage in so-called haemorrhagic laryngitis; in laryngitis sicca with bleeding after separation of crusts; in trauma, abrasions caused by swallowing hard angular bodies in food, surgical operations, and so on. (*h*) Epistaxis with escape of blood into the pharynx. Of all these causes gout is — comparatively speaking — the most fertile source of pharyngeal haemorrhage. Perhaps the greatest difficulty in excluding pulmonary tuberculosis occurs in those influenzal cases in which recurrent haemorrhages from capillary oozing from the fauces, larynx, or trachea are associated with diurnal febrile temperature (see p. 289).

Yet with all these possible sources of haemorrhage from mouth and throat, patients who seek advice for "bleeding from the throat" are generally the subjects of pulmonary haemoptysis. Doubtless the mistake is owing in part to the very prevalent misconception that, unless the blood is coughed up or vomited with food, it cannot come from the lungs or stomach, and, further, because in these cases both doctors and patients are apt to satisfy themselves with explanations more pleasing than the true one, whilst haemorrhage from the throat from all other causes is either very rare or only secondary to graver general affections.

Symptoms.—A capillary oozing from the gums, or from any part of the pharynx, simply gives rise to a taste of blood, and is spat out mixed with saliva. If the oozing of blood occurs during sleep in the recumbent position, the blood may be hawked up with a small quantity of frothy mucus, and so give the impression that it is coughed up from the lungs. On examination, the real source of the haemorrhage may be discovered; but very often this is impossible. If the bleeding be more copious, it may still be possible to examine every part of the upper respiratory and food passages for the bleeding point; but if the blood be poured out too rapidly for any such examination, the head should be held low, so that the blood can run out of the mouth. If it does so without coughing or retching, the source of haemorrhage is almost certainly from the mouth, nose, or throat.

It is more difficult to determine the source of haemorrhage when a patient, without any sign of lung disease, states that a tickling sensation arises in the larynx, and on coughing slightly blood comes in considerable quantity. Of course, if there be evidence of pulmonary disease, or if the blood when coughed up is frothy and bright red, there can be little doubt that it has come from the lungs; but blood which has come from the throat may be bright red, frothy, and mixed with saliva, and on the other hand a pulmonary haemorrhage may be unmixed with air. One point of distinction lies in the fact that in pulmonary haemorrhage the blood may continue to be coughed up with frothy mucus for an hour or two, and then the expectoration generally shews evidence of altered haemoglobin for some days; whereas when blood comes from the mouth or larynx it is soon got rid of completely by coughing and spitting, and, if none is poured out subsequently, all trace of the haemorrhage will disappear in an hour or two. Still, with all these differential signs, it is sometimes extremely difficult to make out the true source of the haemorrhage with certainty.

It is important to remember that tuberculous disease of the lungs often manifests itself by haemoptysis, and that the initial haemorrhage may be considerable without the presence of any physical signs. If, therefore, a patient present himself with a statement that he has had a haemorrhage from the throat, if the pulse rate is persistently increased in frequency, and especially if the temperature is raised at night, then, even though there may be no other evidence of tuberculous disease of the lung, and even if no tubercle bacilli be detected in the expectoration, he should be treated as though the haemorrhage were pulmonary; unless, of course, there be direct evidence that the blood actually came from the throat.

Treatment.—The treatment must be guided by the cause of the haemorrhage. To check the bleeding, the patient may suck ice, and local applications of some astringent, such as tannic and gallic acid powder, or solutions of catechu, matico, or calcium chloride, may be employed; or if the bleeding point can be seen, it may be touched with the galvano-caustic point. These simple methods, together with the other general measures which are usually adopted in haemorrhage, generally suffice to check bleeding from the rupture of small vessels in the mucous membrane of the pharynx or larynx from all causes, if indeed it do not cease spontaneously; but it has sometimes been necessary to ligature the common carotid artery on account of the haemorrhage arising from a suppurating tonsillitis or a malignant growth.

The bleeding having been checked, attention should be directed to the treatment of the underlying cause of the haemorrhage, whether it be a local condition of the throat, or disease of the liver, heart, or kidneys, or a general systemic affection.

Herpes and Pemphigus of the Pharynx (*Herpes, Angina Herpetica*).—We may distinguish two varieties of angina herpetica probably analogous to herpes febrilis and herpes zoster: (*a*) An eruption of vesicles on the soft palate, uvula, or faucial pillars, and rarely on the posterior pharyngeal wall, preceded by and associated with considerable inflammation, redness, and swelling of these regions, and ushered in with rigors or febrile symptoms, the vesicles being either unilateral or irregularly distributed on both sides, and accompanied by smarting pain in the throat, which is aggravated by mastication or swallowing. The vesicles, varying in size from a pin's head to a split pea, are greyish-white and opalescent; they soon burst, leaving white, round, shallow, saucer-shaped ulcers, or, where two or three vesicles coalesce, the resulting ulcer may be correspondingly irregular in size and outline. The herpetic ulcers heal with the subsidence of the pharyngitis. (*b*) An eruption of vesicles, nearly always unilateral, which appear on a patch of erythematous mucosa, unaccompanied by pyrexia, or by any inflammatory condition in the implicated regions, although the vesicles are surrounded by a narrow bright hyperaemic zone. The vesicles may go through the same course as in the inflammatory variety, bursting and leaving a shallow, greyish-white excoriation, or may persist, without alteration, till they spontaneously subside. This condition is unattended by any general disturbance, but causes some pain and discomfort, and is prone to run a tedious course, the vesicles coming and going in long succession. The eruption follows the course of a branch of a sensory nerve, and is probably a local herpes zoster. (*Vide* Fig. 3, Plate III.)

Pemphigus.—Very rarely large bullae occur on the soft palate, uvula, or posterior pharyngeal wall. The bullae very soon rupture and collapse, corresponding patches of white epithelium and whitish thin exudate taking the place of the blebs. Mandelstamm reported five cases, all very chronic, in which the mouth, pharynx, and larynx were affected, one being followed by pemphigus of the skin. Quite recently one of us (F. S.) has seen a case in which pemphigus of the mouth, tongue, and palate was followed by almost universal pemphigus of the skin; and Birkett has observed a case in which the pharyngeal eruption was attended by a bulbous eruption on both forearms.

Treatment is mainly constitutional and should be directed to combating any gouty or rheumatic condition, or to improving the general nervous tone in neurasthenic patients. In cases of chronic recurrent herpes and pemphigus, a course of arsenic should be tried. Locally, if the eruption be painful, applications of cocaine, novocaine, or orthoform will enable the patient to swallow without discomfort, and a simple antiseptic gargle will promote healing of the superficial excoriations following rupture of the vesicles. The general prognosis, however, is extremely serious.

Ulcerative Pharyngitis (*Angina Ulcerosa*).—Superficial ulceration of a faucial pillar unconnected with any specific infective disease occurs somewhat rarely, and though running a mild course is often painful out of all proportion to the local lesion. Heryng first described the condition as *angina ulcerosa benigna.* Usually a single oval superficial ulcer, or excoriation, about half an inch in length, appears on one of the anterior faucial pillars at its upper part, but symmetrical ulcers may occur on both sides or on both the anterior and posterior pillars, greyish in colour, with clean-cut raised edges, with or without a pink margin; the mucous membrane in its neighbourhood being normal or slightly injected or thickened. From a case under one of us (P. W. W.) the coating of the ulcer (Fig. 21) yielded, on culture, streptococci and staphylococci, and Heryng found streptococci in his cases, but it is a moot-point whether these organisms are etiologically related to the ulcers. Dr. Brown Kelly, who has recorded five cases, suggests that the ulcers are herpetic in character and of nervous origin, for such superficial excoriations very closely resemble the abrasion that we should expect after the rupture of large vesicles, and, as Dr. Kelly points out, too much importance must not be attached to the fact that a vesicular stage has not been observed. Freudenthal believes that these ulcers are probably rheumatic in origin, and the observation that some cases clear up rapidly with anti-rheumatic remedies seems to support this contention. The ulcers generally heal without leaving any cicatrix, in the course of a week or ten days.

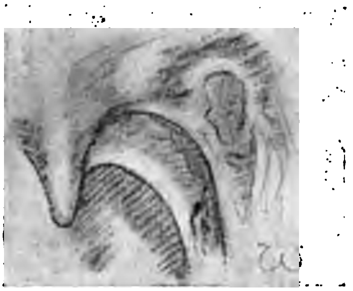

Fig. 21.—Superficial benign ulcer. (P. W. W.)

The *diagnosis* presents little or no difficulty if the ulcer is carefully examined. A commencing diphtheritic patch bears some resemblance to it, but it is covered with a whitish-grey false membrane, whereas the condition under discussion is a grey excoriation. The ulcer might, at first sight, be mistaken for a mucous patch, particularly if the ulcers are bilateral and symmetrical, but the regular sharp edge of the ulcer, and the slightly depressed floor, apart from the history and concomitant circumstances, would hardly leave room for doubt, which in any case would soon be solved with the disappearance of the ulcer.

Treatment by some simple antiseptic pigment or paint is soon followed by the healing of the ulcer. The patient is very often somewhat run down and requires a tonic course, but some cases yield most readily to preparations of salicin.

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Acute Membranous (Non-Diphtheritic) Angina, including Vincent's Angina.—Several forms of infective inflammation of the pharynx and tonsils display a proclivity to be attended with false membrane, or with superficial necrosis simulating a false membrane, and are therefore all more or less liable to be confused with diphtheria, a disease which, although pathologically belonging to this group, has special clinical characters which place it under the domain of general medicine. The formation of pseudo-membrane may be associated with various cocci, the *Bacterium coli* and others, but most usually it appears to be due to Vincent's spirillum and Vincent's fusiform bacillus, and is then designated Vincent's angina. The bacillus is fusiform, slender, beaded, its centre a little thickened with pointed ends, 6 to 12 μ in length, and may be straight, single, or in chains of two or three end to end, is stained by aniline dyes, decolorised by Gram's method, and does not give the Neisser staining reaction. The spirillum or spirochaeta varies greatly in length, thickness, and in number of its spirals, in many respects resembling the *Treponema pallidum*, from which it may be distinguished by the larger size and greater thickness, as well as the "coarser-thread" of the spirals. Like the fusiform bacillus it stains with aniline dyes, though less readily, and is decolorised by Gram. Harwood Yarred and Panton recommended either Loeffler's methylene blue, applied for ten to fifteen minutes, or better still Dudgeon's modification of Leishman's stain, which was introduced by him for staining the *Treponema pallidum*, as by this method the fusiform bacilli are stained blue and their granules purple, while the spirilla are also well stained. Vincent's organisms do not respond to any known culture methods, and soon disappear in cultures, hence no doubt the reason why they are often overlooked in cultures made for suspected diphtheria. The swab should be well rubbed in to the deeper layers of the affected surface, and smears stained and examined forthwith (*vide* also Vol. III. p. 310).

The pellicle or necrotic area is generally found on one tonsil as a single yellowish or greyish-white patch, or it occupies the upper part of a tonsil and the corresponding side of the uvula, with a connecting area running along the velum palati, the tendency to creep over the mucous membrane of the palate and faucial pillars from the tonsils being more marked than in diphtheria. In other cases the patches are found on both tonsils, and there may be multiple patches on the tonsil very suggestive of diphtheria. Again the patches may occupy the margins of the gums around decayed teeth, and corresponding areas of the buccal mucosa may be present. The so-called false membrane is not so tough as in diphtheria; it is thin and soft and friable, is detached with difficulty, leaving an eroded bleeding surface, and often the mucous membranes seem to be definitely ulcerated, in fact there is usually no actual membrane; any similarity to false membrane being due to necrosis of superficial areas of the mucosa. In some cases the whole thickness of the mucous membrane and even the underlying tissues are necrosed, leaving a deep ulcerating surface. The submaxillary cervical glands are generally somewhat enlarged, but they do not suppurate.

The onset of the affection is usually insidious, with loss of appetite, but it may be ushered in with headache, furred tongue, feverishness, and sore throat. In almost every case there is a peculiar offensive odour imparted to the breath by the organisms. The temperature falls about the third or fourth day, and the membrane is generally gone by the eighth or tenth day. But in the more severe cases in which the sloughing is deep, healing may be delayed for several weeks, and some cicatricial contraction may occur. Though usually running a mild course the severe cases may end fatally; thus Bruce, in his account of 10 cases, had two deaths, in one of which the disease spread to the larynx and was followed by bronchopneumonia.

The local treatment consists in painting with some antiseptic, such as iodine in solution. A gargle of boracic acid and permanganate of potassium in solution and probably formamint will be useful.

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Thrush.—This affection is due to the growth of *Oidium albicans* in patches on the mucous membrane of the soft palate, fauces, and mouth, and on the edges and dorsum of the tongue. It usually occurs in poorly-fed, ill-nourished children, but sometimes in cachectic adults, the subjects of some wasting disease. The fungus forms white patches very slightly raised, not surrounded by a hyperaemic zone, though, when the patches are removed, the underlying mucous membrane may be reddened and sore-looking. The child often experiences some difficulty in swallowing from the soreness of the mouth caused by the growth. A simple antiseptic gargle or painting the affected regions with lotio nigra in glycerin or with a solution of salol or borax and honey suffices to get rid of the fungus. (*Vide* also Vol. III. p. 306.)

Pharyngomycosis Leptothricia.—The disease that was, till comparatively recently, described by this name, is now known to be a keratosis, and as such is described below. Nevertheless true mycosis leptothricia does occur, though rarely, a point that has been emphasised by Dr. Brown Kelly. One of us (F. S.), many years ago, recorded a case in a man aged seventy. The soft palate and uvula were much congested, the uvula being oedematous, and on their free surfaces were patches of a milky-white exudation, which could be detached without bleeding, and which the microscope proved to be masses of *Leptothrix buccalis.* The disposing conditions of lowered vitality seem to allow the ever-present leptothrix to develop in patches which are adherent to the surface epithelium, and which, though easily removed without bleeding, or with but slight superficial erosion, and consequent soreness and discomfort, induce slight inflammation in the affected areas. The patches of the fungus are yellowish-white in colour, soft and friable in texture, and appear generally on the uvula and soft palate, but frequently on the mucous membrane of the pharynx, larynx, naso-pharynx, etc. They quickly yield to gargles of chlorate of potassium, or applications of argyrol, protargol, or mercurial or other antiseptic solutions.

Keratosis Pharyngis.[1]—Formerly the leptothrix fungus and spores which are almost invariably present in the concretions of tartar round the teeth and on the papillae of a coated tongue, and which are very frequently found in the crypts of the tonsils, were supposed to be the active factor in this condition. They take root in the tissue and germinate, forming characteristic milky-white, chalk-like outgrowths. But it has been shewn by Siebenmann and others that the organisms which flourish in the fringes and borders of the epithelial layers and often accumulate on the horny outgrowths to such an extent as practically almost to compose the most projecting portion, are merely saprophytic, and have no action in causing the affection. Wherever there are lymphoid follicles or tonsillar crypts the invaginated epithelium may shew the characteristic changes, the abnormal proliferation of keratinised epithelial cells, dense layers which either fill the crypts or project from the surface as milky-white, chalk-like outgrowths. A noteworthy point, emphasised by Dr. Brown Kelly, is that the condition rarely occurs outside Waldeyer's ring, *i.e.* the ring formed by the pharyngeal, faucial, and lingual tonsils, and their connecting tracts of adenoid tissue.

The horny projections really grow from the bottom of the crypts and acinous glands, and are most frequently seen on the palatine and lingual tonsils; though the soft palate and uvula and posterior pharyngeal wall also are often the seat of the growth. The mucous membrane around the growths of the fungus is healthy, unless slightly irritated and inflamed from the invading organisms of the mouth, but the masses are remarkably adherent and often cannot be torn away without some of the epithelium of the matrix. Sometimes they are soft and break off short when removal is attempted.

The affection generally occurs in patients who are run down in health from one cause or another, and is especially apt to follow digestive disorders.

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[1] We have adopted the name suggested by Dr. Brown Kelly, as preferable to hyperkeratosis lacunaris proposed by Siebenmann, for the affection formerly known and described by us in the previous edition as pharyngomycosis leptothricia.

Symptoms are generally very slight or altogether absent, and often enough the patches are accidentally discovered by the patients. A certain degree of discomfort, stiffness, and dryness may occasionally be felt in the throat; whilst in some cases there is an irritating cough, and the voice may be impaired. It is very doubtful, however, how much even of these slight symptoms is directly due to the growths, and how much to the dyspeptic troubles and impaired health with which the affection is generally associated.

Diagnosis.—The only affection that may be confused with this keratosis is chronic lacunar tonsillitis with yellow caseous exudation in the crypts. The yellow masses, however, are readily extruded; whereas keratotic masses are very adherent and are chalk-white in colour. The absence of pain, febrile temperature, and constitutional disturbance at once distinguish the affection from acute tonsillitis or diphtheria.

The *treatment* should be directed to improvement of the general health. Many forms of local treatment have been advocated, but even when most vigorously and perseveringly carried out they are all very tedious, and fail to prevent the return of the condition; whereas we have almost always found that with improved health the growth disappears spontaneously. Thus, in our opinion, local treatment is hardly ever required.

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Actinomycosis.—A primary actinomycotic infection of the pharynx and tonsils is exceedingly rare, or, at any rate, has rarely been recognised. Jonathan Wright found one case of actinomycosis of the tonsil in the course of histological examinations of seventy-five hypertrophied tonsils. The tonsil had been removed from a boy, aged twelve, and contained, what at first appeared to be an ordinary abscess cavity, but which Wright proved to be in reality a distended crypt filled with the broken-down epithelium and the ray fungus. This author refers to four somewhat similar cases of actinomycosis of the tonsil, described by Ruge in 1896, and another by Lesin, in which a local lesion existed in the tonsil, the patient having succumbed from cerebral infection. When the mouth is affected it is generally the gums around carious teeth that are attacked first, but nodules may appear on the tongue or pharynx; when these ulcerate, the viscid discharge will be found to contain the specific fungus (*vide* "Pathology of Streptothrix Infections," Vol. II. Part I. p. 302, and "Actinomycosis," Vol. II. Part I. p. 324).

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Glanders.—A full description of the disease is given in Vol. II. Part I. p. 201, and the nasal manifestations are described on p. 56. In acute glanders the nasal infection may spread to the naso-pharynx, and invade the soft palate and pharynx, and even extend to the larynx causing oedematous laryngitis, and to the lungs and pleurae. The patient usually becomes more and more prostrate; delirium supervenes and passes into coma, and death follows in one or two weeks. For the diagnosis and treatment the reader should refer to Prof. Woodhead's article in Vol. II. Part I. p. 201.

Tuberculosis of the Pharynx.—*Etiology.*—The immediate and remote causes of tuberculous disease of the fauces and pharynx are the same as in pulmonary tuberculous disease, to which the pharyngeal affection, which is one of the rarest manifestations of tuberculosis, is almost invariably secondary. Occasionally the pharyngeal affection appears to precede or to appear simultaneously with pulmonary tuberculosis; but if we except the tonsils primary pharyngeal tuberculosis is very rare. Chronic enlargement of the faucial or pharyngeal tonsils disposes those structures to tuberculous infection; but no definite reason can be assigned for the occurrence of the disease in the soft palate.

Pathology.—Pharyngeal tuberculosis may be either acute or chronic. Only two or three cases are recorded in which the acute form was believed to be primary; the chronic variety is more frequently unaccompanied by evidence of pulmonary infection. The route by which the bacilli gain access to the infected tissues is not at present known. The old view that the pharyngeal tissue is directly infected by the sputum does not account for the fact that the deeper tissues are affected first; and that the superficial ulceration arises by the extension and breaking down of deeper-lying miliary tubercles. On the other hand, the tendency for tuberculous disease in this region to attack either the anterior surface of the soft palate, the posterior pharyngeal wall, or the laryngo-pharynx opposite the cricoid ring, suggests that slight or superficial abrasions produced in swallowing food provide the portal for the entrance of infection, which in the case of the tonsils is always present in the crypts.

The tonsils are much more frequently affected than formerly believed; and these glands are in many cases to be held responsible for the entrance of tubercle bacilli, as indeed of other microbes also into the system. Krueckmann has shewn that tuberculosis of the cervical lymphatic glands almost always depends upon the invasion of the glands by way of the tonsils; and in no less than 60 per cent of cases of tuberculosis of the lungs examined on the post-mortem table by this observer, tubercles were detected in the tonsils: similar results had previously been obtained by Strassmann and Dmochowski. Many cases of pharyngeal adenoids have been proved to be tuberculous; in some giant-cells have been demonstrated, whilst a very large proportion contain tubercle bacilli. Masked tuberculous disease of the tonsils undoubtedly occurs in the course of pulmonary tuberculosis; but it is probable that a similar condition of the tonsils often precedes the establishment of the lung affection.

The subsequent course of the tuberculous deposit differs in no respect from tuberculous disease in other regions; caseation and breaking down soon result in characteristic ulceration.

The *symptoms* of tuberculosis of the pharyngeal mucous membrane and of the tonsils differ in several respects; though many of the symptoms are common to all tuberculous processes.

The acute form of the pharyngeal affection, due to secondary infection through the lymphatics, usually begins with pain in the faucial region, which on examination is found to be hyperaemic and slightly swollen. The soft palate, if the seat of deposit, becomes stiff and paretic; and in the course of a day or two several discrete, muddy-grey miliary tubercles are visible, slightly elevated, but obviously below the translucent mucous membrane. The initial hyperaemia gives place to a more or less general anaemia of the soft palate, as the tubercles increase in number and coalesce. Very soon discrete or confluent ulceration of the tubercles occurs, and by superficial extension the originally small solitary ulcers coalesce and form a larger superficial ulcer covered with greyish-white, diffluent, breaking-down, caseating matter, and with irregular "worm-eaten" or "mouse-nibbled" margins which are flush with the surrounding mucous membrane. Fresh tubercles meanwhile appear, only to pass through similarly rapid phases of development.

Ere this the infiltration of the soft palate has resulted in failure of its functions; consequently the voice is nasal, and fluids escape by the nose on drinking. Deglutition becomes extremely painful, and coughing almost impossible; consequently the patient is unable to get rid of the copious, sticky, stringy, muco-purulent discharges covering the parts, which accumulate and dribble from the open mouth, or are expelled by feeble attempts at hawking. As in acute miliary tuberculosis of the lungs, the temperature ranges high, without presenting the hectic character; but the emaciation and general prostration are more rapid.

In the more common chronic form, which is probably due to a direct and superficial inoculation, the formation of tubercles is less obvious, the ulceration is indolent, whilst granulations and nodular thickening may cause it to resemble lupus. Pain, wasting, and febrile symptoms are well marked; though, of course, concomitant pulmonary disease will be attended by the usual clinical phenomena.

Tuberculous disease of the tonsils occurs alone or in association with the palatine deposit. It manifests itself by congestion and enlargement of the glands, and superficial ulceration soon occurs, the ulcers being multiple and with irregular ill-defined margins; they are covered with greyish-white muco-purulent matter which contains the specific bacillus.

The *diagnosis* of the acute form has to be made from diphtheria, follicular tonsillitis, syphilis, herpes, and small-pox; whilst the chronic variety must be distinguished from lupus and syphilis.

The presence of pulmonary lesions will, of course, at once suggest the probable nature of the throat affection, and the characteristic miliary tubercles and superficial "worm-eaten" ulceration will serve to exclude diphtheria, syphilis, and small-pox: the general symptoms will likewise differ from those of small-pox and diphtheria. In herpes of the fauces, the clear vesicles and absence of severe constitutional disturbance should prevent any mistake in diagnosis. In lupus, apart from the rarity of the primary faucial cases, the occurrence of slowly-forming, clear, apple-jelly-like, painless tubercles and the tendency to cicatrisation of the clean ulcers should serve to distinguish it from the irregular ulcers of tubercle, which are covered with detritus and never cicatrise.

Treatment.—In all cases the affected tissues, having been cocainised, should be thoroughly scraped with a sharp curette, and pure lactic acid applied daily. Occasionally the disease may be arrested, at least temporarily, by this method. In the acute form ice should be sucked and the throat frequently sprayed with a solution of cocaine (4 per cent) and menthol (20 per cent) in adepsine oil; or orthoform or anaesthesine should be insufflated once or twice daily for the relief of the pain and dysphagia.

The general treatment should be the same as in pulmonary tuberculosis.

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Syphilis of the Pharynx.—Syphilitic disease may affect any part of the fauces and pharynx, and in the more exposed regions occurs in all stages—namely, (1) Primary chancre; (2) Erythema; (3) Mucous patch (condyloma); (4) Superficial ulceration; (5) Gumma; (6) Deep ulceration; (7) Cicatrix. Though it is generally possible to assign pharyngeal syphilis to the so-called secondary or tertiary periods, the statements made on this point in reference to laryngeal syphilis (see p. 209) apply, but to a less extent, here.

(1) The primary sore, though decidedly rare in this region, has been observed in a good many cases, chiefly on the tonsils, very occasionally on the faucial pillars; for whereas the irregular surface and crypts of the tonsil form a ready means of entrance for the infection, the smooth

unbroken surface of the fauces and soft palate afford but slight opportunity for inoculation; consequently, with very few exceptions, the essentially localised initial sore is encountered on the tonsils only, and generally in cases in which the tonsils were already chronically enlarged.

The affected tonsil is red, and the sore is generally eroded, without marked ulceration, presenting a sharply-cut, well-defined margin, with a small amount of sticky, greyish-white secretion covering the floor of the ulcer. There is very well marked induration on palpation, often stony hardness. The sore commonly extends over the whole surface of the tonsil, and the submaxillary glands are very much enlarged and tender to pressure; but they do not suppurate. Pain is seldom well marked and is often absent; yet in some instances it is severe and lancinating in character.

(2) Erythema usually occurs between six weeks and four months after the initial sore, and is generally coincident with cutaneous erythema or the papular syphilide. It presents a peculiar, almost characteristic bright bluish-red, symmetrical hyperaemia, generally confined to the soft palate and pillars of the fauces, rarely implicating the tonsils, with a somewhat sharply-defined border, so that the line of demarcation between the hyperaemia and normal mucous membrane is almost abrupt. This appearance should always lead to the suspicion of syphilis.

There are generally no symptoms sufficiently notable to attract the attention of the patient; some stiffness of the parts may be observed.

(3) Mucous patches usually appear about the fourth month after inoculation, but they may be observed some years after, as in a good many cases they come and go for long periods. Sometimes they appear while the primary sore is still present. Whilst ordinarily coexisting with a papular cutaneous syphilide, they often appear when there is no general manifestation of the disease. They are usually more or less bilaterally symmetrical, slightly elevated, bluish-white, and are situated on the fauces, tonsils, or posterior wall of the pharynx, whilst similar patches are frequently present on the inside of the lips, the gums, the inside of the cheeks and the tongue; they are attended with slight congestion of their neighbourhood and may lead to superficial abrasion.

(4) Superficial ulceration is especially prone to occur on both tonsils, forming remarkable symmetrical kidney-shaped ulcers, with a greyish-white, ill-defined border. But the ulceration may be limited to the posterior surface of the soft palate and the rhino-pharyngeal space. It is one of the earliest manifestations of secondary syphilis, often preceding or accompanying the cutaneous erythema, and, like the latter, usually disappears very soon, without sore throat. On the other hand, it may persist and be followed by a more painful inflammatory sore throat.

(5) Gumma is generally single, and in the soft palate, pillars of the fauces, tonsils, and particularly in the posterior pharyngeal wall may

appear as a smooth, uneven, red or angry-looking swelling, covered and surrounded by congested mucous membrane situated usually in or near the middle line. It rarely gives rise to much pain, and frequently to none whatever; but a sense of fulness and discomfort in the part or a difficulty in deglutition first attracts the notice of the patient. Very soon its centre becomes yellowish and soft, and, pain being absent, the gumma often breaks down before the patient consults a medical man, when a typical deep crateriform ulcer, with steep margins and a base covered with sticky mucopus and debris, has already been formed.

(6) Tertiary syphilitic ulceration is always due to the disintegration of gummatous deposit. In the earlier cases these ulcers are most often found in the soft palate, faucial pillars, or uvula; but tertiary ulceration occurring many years after the initial lesion more frequently affects the tonsils and posterior pharyngeal wall. A gumma may also form on the posterior wall of the soft palate or in the naso-pharynx. In the former case very rapid perforation of the palate or dropping off of the uvula may occur if the actual condition has not been diagnosed and treated. In view of the frequently painless character of the affection it is therefore essential that the posterior surface of the soft palate should be inspected by the rhinoscope, especially when the anterior surface appears red and infiltrated. Not only may the soft palate and uvula completely disappear, but the destructive process may invade the hard palate and open into the nasal passages. Occasionally these deep ulcerations are attended with serious haemorrhage (Fig. 1, Plate IV.).

(7) Cicatrix.—Deep syphilitic ulceration is generally followed by contraction, distortion, and adhesion of the tissues involved: thus, the soft palate may be bound down to the posterior pharyngeal wall, more or less completely shutting off the rhino-pharyngeal space; or the uvula may become adherent to the faucial pillars. Syphilitic scars may often be recognised by their stellate or radiating appearance due to the contraction and dragging of neighbouring tissues towards the former site of the ulcer as a centre. A similar process occurring in the lower pharynx or oesophagus may lead to obstruction to the passage of food.

Inherited Syphilis.—Inherited syphilis affecting the pharynx generally manifests itself in early infancy or at the age of puberty. It may assume the form either of secondary or of tertiary lesions, of syphilitic catarrh, erythema and superficial ulceration, or gummatous deposit with deep ulceration. Syphilitic catarrh and superficial ulceration are generally associated with a similar condition in the nasal passages, giving rise to what is commonly called "snuffles." Deep ulceration of the fauces or pharynx is very frequently combined with destructive ulceration of the nasal bones, as has already been described as a consequence of tertiary manifestations in acquired syphilis.

Diagnosis.—A tonsillar chancre is liable to be mistaken for tertiary ulceration, carcinoma, or tuberculous disease. From tertiary ulceration it is distinguished by its superficial character, the stony hardness of the tonsil, and the large cervical bubo; whilst the early appearance (in from two to four weeks) of secondary cutaneous manifestations will always settle the question. It is less easy to distinguish between a chancre and carcinoma, and very often it is impossible to do so from mere inspection till other syphilitic phenomena arise; but the duration of the affection, and the fact that the margins of the ulcers are flush with the surrounding tissues, which in turn are congested, will favour the diagnosis of chancre. The patient's age must also be taken into consideration. Malignant disease of the pharynx hardly ever appears before the age of thirty-five. The effects of mercurial treatment, and, finally, the microscopical examination of a fragment of the ulcerating tumefaction, will assist us in arriving at a definite diagnosis. Tuberculous ulcers are more irregular, they present a mouse-nibbled appearance, they are covered with copious sticky mucopus, and are usually multiple. The enlargement of the cervical glands is less rapidly developed, and the evening rise of temperature and increased frequency of the pulse, even in the absence of concomitant pulmonary lesions, should lead to an examination of the debris for the specific bacilli. In its initial stages the disease may be mistaken for a commencing tonsillitis, but the development of the sore will quickly settle the diagnosis.

Mucous patches and condylomas may be taken for diphtheria, from which the absence of constitutional symptoms and the presence of coexistent syphilitic skin disease should distinguish them. As a rule, the bluish-white, symmetrical, opalescent appearance of the mucous patches surrounded by apparently healthy mucous membrane is in itself sufficiently characteristic. From Vincent's angina they may be distinguished by their tendency to bilateral symmetry; the bluish-white colour of mucous patches are more translucent than the yellowish-white opaque necrotic areas of Vincent's angina; mucous patches cannot be detached, whereas the patches of Vincent's angina are fairly readily removed. Moreover, the coexistence of similar patches on the lips, etc., and of papular cutaneous syphilides will generally serve to remove any doubt that may arise.

A gumma is sometimes diagnosed as quinsy, especially when its formation is attended with pain and febrile symptoms; or it may be mistaken for a fibroma, sarcoma, or carcinoma. In the former case the facts that it is unilateral, little tender to pressure, not painful, and not inflamed on the mucous surface, favour the diagnosis of gumma. Fibroma is very rare, but in doubtful cases antisyphilitic treatment must be relied upon to distinguish both this and malignant growths from gumma. Sarcoma is less rapid in its growth, and presents a more highly coloured and succulent aspect, but the deep ulcer with foul-smelling disintegrated debris sometimes closely simulates a breaking-down carcinoma, from which it is distinguished by the red areola surrounding the margin, by the edge of the ulcer not being raised, and by the absence of the fungating base. In doubtful cases a microscopical examination of a

PLATE IV.

TERTIARY SYPHILIS OF THE PHARYNX.

Fig. 1. A gumma of the soft palate has broken down into a characteristic deep crateriform ulcer with sharply defined undermined edges, around which is a fairly defined deep red areola, and dirty yellowish débris are seen at the bottom of the ulcer. (Case of P. W. W.)

" 2. An atypical form of tertiary syphilis of the posterior pharyngeal wall. Fairly circumscribed, somewhat superficial ulcerations are present, resembling in some respects tuberculous ulceration. But the ulcers are more defined, and the unbroken bands of mucous membrane are brighter in colour than in tuberculous disease. The whole condition rapidly cleared up under a course of iodide of potassium. (Case of P. W. W.)

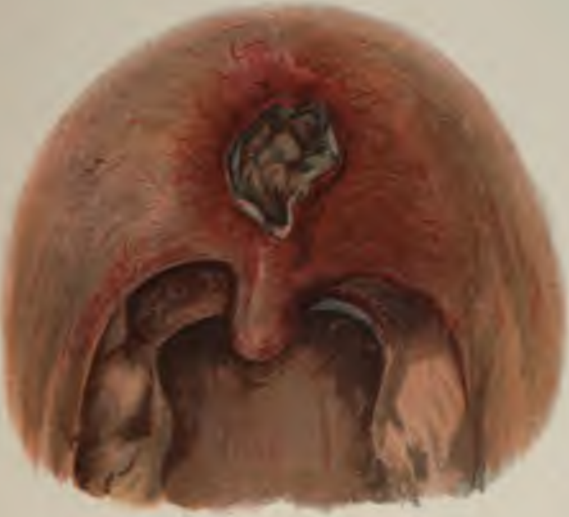

Fig. 1.

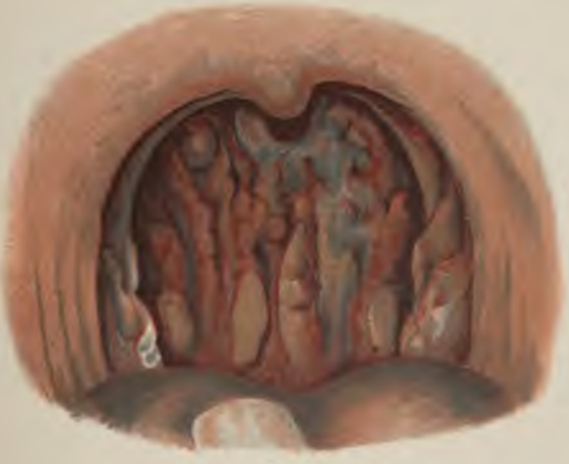

Fig. 2.

fragment, and rapid diminution in size of the "growth" under iodide of potassium, would probably reveal the true nature of the case (*vide* Fig. 2, Plate IV.).

Whilst, as a rule, the symptoms of syphilitic disease of the upper air-passages are well characterised, and their diagnosis is not particularly difficult, occasionally cases of this affection are met with, which do not at all correspond to the usual notions. Thus, in some instances manifestations of tertiary syphilis appear at an unusually early period, occasionally even within the first year from the primary infection. In other cases very violent pain is complained of, whilst ordinarily that symptom is either absent or its degree insignificant. Again, in other cases the appearances of the infiltration or ulceration and their situation are quite different from the typical aspects, and may be clinically indistinguishable from other forms of infiltration, particularly from those due to invasion of pathogenetic organisms, such as the pneumococcus. Finally, in some cases of what has justly been called malignant syphilis, the regular antisyphilitic remedies, mercury and iodine preparations, instead of, as is usual, exercising an almost miraculous curative influence upon the disease, actually aggravate its course. It is impossible to discuss here at length all the varieties that may be met with, and we must refer the reader to the collections of such unusual cases, published by one of us (F. S.) at various times, in which suggestions will also be found as to the treatment of rebellious cases.

Treatment.—Should the *Treponema pallidum* (*Spirochaeta pallida*) prove to be the cause of the syphilis, its presence in the affected tissue would, of course, settle the diagnosis in doubtful cases. Mucous patches which do not disappear with antisyphilitic remedies may be painted at intervals with a solution of nitrate of silver (20 grains to the ounce). This, however, will be very rarely required. Superficial ulcers may be painted with solution of chromic acid (gr. x. ad ℥i.); the ulcerated surface must previously be wiped dry. Deep ulcers should be cleaned by a simple alkaline gargle or spray, and a mercurial antiseptic gargle used afterwards. Cicatricial stenosis of the rhino-pharynx may require division with subsequent dilatation persistently repeated for a long time, as all syphilitic scars are apt to contract afresh.

The general treatment of syphilitic disease of the pharynx does not differ from the treatment of similar manifestations in the larynx, to which the reader is referred (p. 214). The remarks made there on the necessity for avoiding too rigid adherence to any routine method of treating secondary lesions with mercury and tertiary lesions with iodide of potassium, apply with equal force to syphilitic disease of the pharynx. In some obstinate cases of malignant syphilis of the throat sarsaparilla will be found useful, when neither mercurial nor iodine preparations can be taken.

Local treatment is usually unnecessary; but in all syphilitic affections of the pharynx an antiseptic gargle, such as a solution of perchloride of mercury (1 in 1000), may be used with advantage.

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Scleroma.—Formerly known as "rhino"-scleroma, the disease is now proved to affect other regions of the body also, and though most frequently the nasal passages are its seat, it often attacks the larynx and pharynx, and sometimes the trachea (in one case the upper eyelids). There is no doubt that, at any rate, many, and probably most, of the cases hitherto described as chorditis vocalis inferior which were met with in some parts of Germany, were in reality of the nature of scleroma, and the view of Pieniaczek that Stoerk's blennorrhoea must be considered as scleroma, has been endorsed by Gerber and others. The majority of cases have been reported in East Prussia, and the south-east of Europe, and a few in America. The first case demonstrated in this country was shewn by one of us (F. S.) in conjunction with Dr. Payne in 1884; other cases have been recorded and demonstrated by Dr. Dundas Grant in 1899 and by Dr. St. Clair Thomson in 1907.

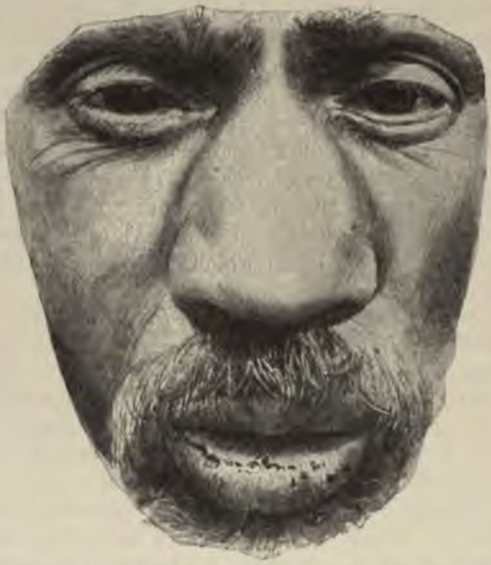


FIG. 22.—External Nose. Nasal distension of cartilaginous hardness beneath unaltered skin. (Russian case from the Province of Kowno. Clinic Gerber.)

The disease is one of the infective granulomas, characterised by nodular thickenings of the mucous membrane of the nose and throat or of

PLATE V.

SCLEROMA OF THE PHARYNX, NOSE, AND LARYNX.

Figs. 1 to 6. (Gerber.)

Fig. 7. Drawing of a case of laryngeal scleroma. (Smurthwaite.)

PLATE V.

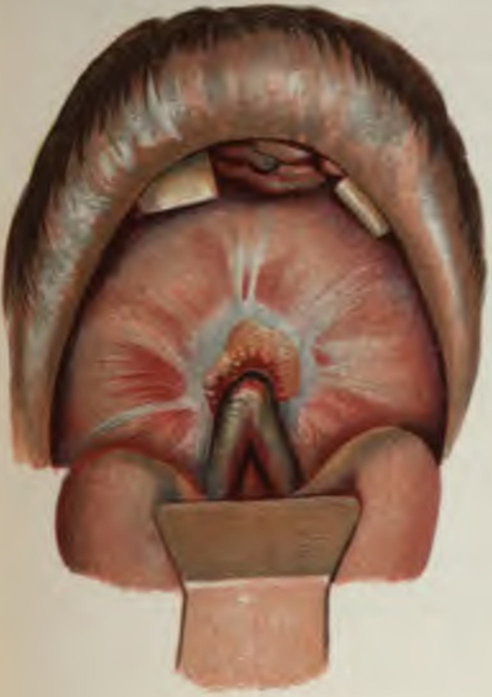
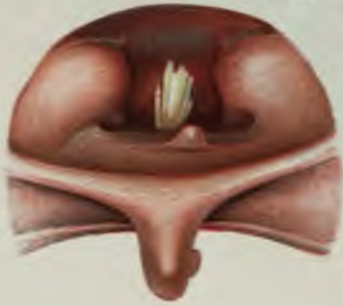


Fig. 1.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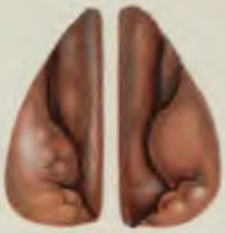
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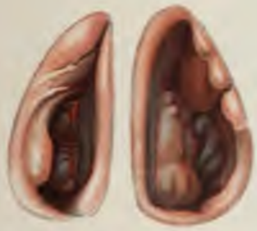
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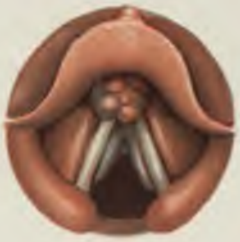
Fig. 6.

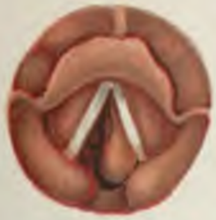
Fig. 3.

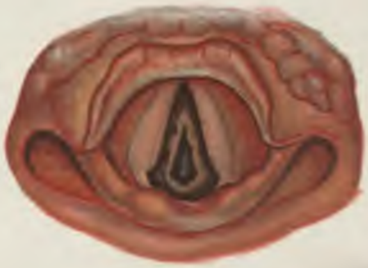
Fig. 7.

the nose externally. Although apparently there is no definite proof of their being so, the short, thick, blunt-ended bacilli found in the Mikulicz bodies, first described by Frisch, are presumably the essential infecting agents.

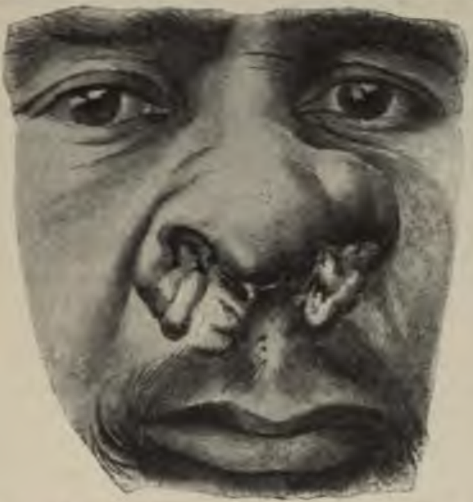

FIG. 23.—Distension of the nose by enormous multiple nodes extending to the upper lip. (Russian case from the Province of Volhynia. Wolkowitsch.)

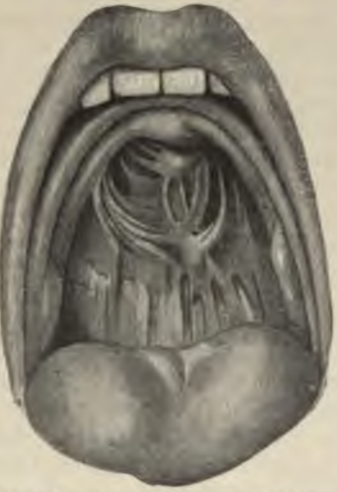

FIG. 24.—Pharyngeal aspect. Two nodes on the posterior wall of the pharynx. Considerable formation of folds between them and the palatal arches, which, together with the uvula, have become adherent to the posterior wall of the pharynx. (Vienna Laryngological Clinic. Juffinger.)

The substance of the swelling is mainly composed of cells of two varieties: (*a*) The large plasma-cells with homogeneous protoplasm which stains only slightly, and with a nucleus, when present, at the border of the cell. The cells generally contain several of the bacilli grouped about the periphery of the cell. These are the cells described by Mikulicz as characteristic of scleroma. (*b*) The hyaline cells, varying in character according to the stage in the formation of hyaline matter occurring singly and in small collections. Whether the hyaline cells bear any direct relation to the large plasma-cells, or merely correspond to the hyaline degenerated cells of other chronic inflammations of mucous membrane is as yet undetermined.

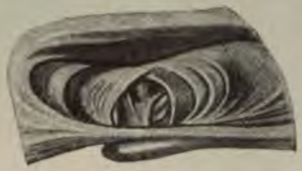
FIG. 25.—Posterior rhinoscopy. Centripetal narrowing of the nasopharyngeal cavity caused by cicatricial membranes. Uvula fixed by a cicatricial process to the lower margin of the soft palate.

Symptoms.—The onset of the disease is usually most indefinite and its very slow progress, often gradually increasing for years and then remaining stationary for years, before again shewing evidence of its development, very frequently leaves the patient in doubt as to the date of its earliest manifestations. He usually complains of stuffiness in the nose, or of actual nasal obstruction, or of persistent hoarseness. If the larynx is affected, sticky secretions and crusts, resembling and sometimes mistaken for ozaena, are usually present, and as the disease advances, nasal occlusion or laryngeal obstruction, with dyspnoea and hoarseness or aphonia. When the skin of the nose or other parts is affected, a sense of stiffness and discomfort in the infiltrated integument arises with the progressive development of the nodules, deformity and marked facial disfigurement appears (*vide* p. 54, and Plate V, and Figs. 22, 23).

The nodules in the mucosa may be soft but are usually firm, and sometimes of almost ivory hardness. They are at first pink or greyish-pink, but as they get firm are more usually greyish-white. They are not prone to ulcerate or break down, but the surface is often covered with crusts of secretion, which on removal may leave a bleeding surface.

In the larynx the disease may occur as the primary infection, and is usually bilateral and infraglottic, though in other cases it appears above the glottic aperture. On the ventricular bands or immediately below the vocal cords the diffuse infiltrations form pink or greyish, protruding, flat, granulomatous swellings. Scleroma usually begins anteriorly, gradually spreading back to the region of the processus vocales. Occasionally it is unilateral, and thick laryngeal diaphragms may form in the course of years. It is also seen in the base of the epiglottis. Less rarely the trachea becomes affected by extension downwards from the disease in the larynx; in addition to the collections of secretion in the trachea, the thickening and contraction may cause serious dyspnoea and stridor. Some instances of primary infection of the trachea are recorded.

In the pharynx it forms cicatricial contraction of the faucial isthmus, or contraction of the uvula and soft palate, enlargement of the tonsils, or the tongue may be involved and shrink. In the rhino-pharynx thickening of the vomer, enlargement of the Eustachian cushions, and in the nasal passages thick flat infiltrating nodules slowly appear.

There is reason to believe that the disease is contagious, but probably only in the way that leprosy is communicable. Probably some local morbid process disposes to, and is necessary for the infection to take place. English readers will find a case described, together with a good summary of the literature of laryngeal scleroma, by Emil Mayer (4). The illustrations (Plate V. Figs. 1-6) kindly furnished by Prof. Gerber serve to demonstrate the characteristic appearances of the disease.

Treatment is so far very unsuccessful. For the laryngeal stenosis intubation has been tried in two cases with some success and the external conditions have been benefited by the use of *x*-rays.

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Gouty Affections of the Throat.—The so-called lithaemic diathesis is a much more frequent cause of throat disease than is generally believed, the throat being often affected in patients who present no definite evidence of gout, or who have never had any acute joint inflammation.

Symptoms.—The throat manifestations of gout may be acute or chronic.

Acute gouty pharyngitis, tonsillitis, or laryngitis may result from exposure to cold, or may occur without any obvious local cause in predisposed persons. The affection may run the usual course of acute inflammation of these regions, or may yield abruptly to an ordinary attack of acute gout. One of us (W. W.) observed a case of a medical man, who had had many definite attacks of gout, in which nocturnal laryngeal spasms were prone to occur whenever an error in diet rendered the patient gouty. A similar case was observed by Sir Clifford Allbutt; in this case, in a fine and vigorous but gouty man of middle age, the spasms, which recurred at intervals for some years, would compel the sufferer to spring from his bed in an agony: the local signs were never very notable. The chief distinguishing subjective symptom is that the pain or spasm is out of proportion to the degree of inflammation.

Objectively the fauces or larynx, as the case may be, are acutely inflamed and bright red, the inflammation, as a rule, being strikingly patchy in appearance; the inflammation is particularly noticeable on the lateral pharyngeal walls, whilst the uvula may be oedematous. Although often enough there is nothing in any way characteristic in the appearances presented, one of the most usual features is the red glazed surface of the mucous membrane, which Sir D. Duckworth compared to mucous membrane freshly brushed over with glycerin. The uvula is sometimes not only swollen but thick and elongated. In rare instances local acute gouty inflammation arises almost suddenly in the fauces or tonsil, with a dark red oedematous appearance, more diffused than in peritonsillitis, without exudation or enlargement of the neighbouring cervical glands. These conditions were observed by Lermoyez and Gasne in a patient in whom all the throat manifestations disappeared as abruptly as they arose when they gave place to a typical attack of podagra.

The more chronic form may be indistinguishable from ordinary pharyngitis and laryngitis; but in most cases there is marked thickening of the lateral walls of the pharynx, two symmetrical bands of swollen mucous membrane passing in a vertical direction along the angle between the posterior and lateral walls of the pharynx.

As indicative of gout we lay particular stress on lateral pharyngitis with a sense of uneasiness or pain on swallowing; the pain may be of a darting character and shoot up to the ears. Small tophi may form under the mucous membrane and may be expelled; or urate of sodium may be discharged from accumulations in the mucous membrane. These gouty concretions, in exceedingly rare instances, may form on a vocal cord (as in a case recorded by Virchow), or within the crico-arytaenoid joint, causing ankylosis. A gouty inflammation of such character may produce symptoms and physical signs indistinguishable from those of laryngeal cancer. In a case seen by one of us (F. S.) in consultation, thyrotomy was performed on the suspicion that there was malignant disease of the larynx; but the supposed new growth turned out to be a gouty concretion embedded in a vocal cord. A similar instance came before Krishaber and Morell Mackenzie; in this case the laryngeal disease disappeared while the patient was undergoing treatment for gout. De Mussy described a case in which small concretions of carbonate and urate of sodium appeared from time to time on the posterior pharyngeal wall.

The diagnosis of gouty affections of the throat is often simple enough if the peculiar patchy aspect of the inflammation and the lateral pharyngitis are noted. Such appearances, especially when attended with unusual sensitiveness and pain in the throat, should lead to inquiry into the family and personal history of the patient, and to careful investigation into any constitutional or other local manifestations of the gouty habit; in many instances, however, the diagnosis must largely depend on the response to suitable treatment. On the other hand, it does not by any means necessarily follow that every inflammatory affection of the throat in a gouty patient is itself of gouty nature.

The treatment is simply that suited for systemic gout, the only local treatment necessary being some sedative spray, or pastille containing menthol and cocaine. Tincture or wine of colchicum (♏ x to xxx), with or without bicarbonate of potassium or salicylate of sodium, may be added to a tumbler of Vichy water and taken twice daily after meals; in the more chronic cases a visit to some appropriate spa, Harrogate, Bath, Strathpeffer, Carlsbad, Marienbad, Kissingen, Vichy, Contrexéville, etc. is highly to be recommended to patients of sufficient means and leisure. Aspirin, in the form of tablets (two of 5 grains each twice daily), will often be found very useful. The more acute cases should be treated as acute attacks of gout, and in the usual manner, the patients being confined to the house. In any circumstances the larynx, if inflamed, should be rested as much as possible, and all sources of irritation removed; and, after the gouty condition has been combated by appropriate treatment, the treatment suited to subacute or chronic inflammation of the pharynx or larynx may be necessary. Needless to say, the usual dietetic rules for gout must be strictly carried out.

Rheumatic Affections of the Throat.—The causes of rheumatic affections of the throat differ in no respect from those of rheumatic affections occurring in other parts of the body; nor can it be said that there are any distinguishing characteristics of rheumatic pharyngitis, tonsillitis, or laryngitis. The very intimate pathological connexion between acute lacunar tonsillitis, peritonsillitis, and acute rheumatism is now widely recognised; but it is important to remember that a large proportion of cases both of acute and chronic pharyngitis and laryngitis is of rheumatic origin, for success in their treatment will very much depend on a correct diagnosis. Pain, stiffness, and inflammation of the fauces very frequently precede an attack of acute rheumatism, and either subside or are disregarded when the acute joint symptoms are manifested. In other cases the throat symptoms persist for days or weeks without further development, and not seldom recur regularly whenever the patient is exposed to cold or damp. Rheumatic inflammation may arise in and around the crico-arytaenoid joints, or directly attack the intrinsic muscles and peripheral nerves, causing diffuse neuritis, impairment of mobility of one or both vocal cords, and in some cases marked tenderness to pressure. The diagnosis of "rheumatic" paralysis of the vocal cords, however, ought not to be made until all other possible *organic* causes of the palsy have been excluded.

Treatment.—It is unnecessary to suggest the general treatment to be adopted in rheumatic affections of the throat, for it is simply that suited to rheumatic diseases of the joints. Locally a sedative spray, such as menthol and cocaine in colourless oil of vaseline, and other local treatment referred to in the sections on acute and chronic inflammation of the pharynx, tonsil, and larynx, should be carried out.

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NEW GROWTHS OF THE PHARYNX AND TONSILS. — A. **Benign Neoplasms.**—It is as little possible in the present state of knowledge to assign any definite cause for the appearance of benign neoplasms in the fauces as elsewhere, with the sole exception of dermoid tumours, which are very rare and represent abnormalities of development.

Benign growths are not frequent in the fauces, though repeated demonstrations at the late Laryngological Society of London have shewn that the tonsils are more often the seat of innocent growths than had been formerly believed. *Papilloma* is by far the most common form; the small, warty, sessile, or pedunculated, light-pink growths, with cauliflower or granular surface, being usually attached to the margin of the soft palate, the pillars of the fauces, or the uvula. Next in point of frequency comes the *adenoma*, a hard, rounded, sessile tumour of slow growth, covered with smooth, irregularly rounded mucous membrane of normal appearance, arising in the mucous membrane of the anterior or posterior surface of the palate or in the tonsil, and often attaining a considerable size. *Fibroma* is very rarely met with in the fauces, and is more common in the rhino-pharynx, where as a rule it is attached to the vault of the pharynx. These tumours are somewhat rapid in their growth, and may become as large as a hen's egg or a small orange. They are hard, rounded, smooth and red on the surface, and sometimes highly vascular. *Angioma* occurs as a purple, nodular, soft, vascular growth, composed of enlarged tortuous blood-vessels held together by a small amount of connective tissue. *Calcareous* concretions are rather frequently present in the tonsil, more rarely in the soft palate. Being covered by mucous membrane, the swelling may simulate a growth (*vide* p. 174).

Diagnosis.—Papilloma often bears a very strong resemblance to a warty epithelioma. There is generally no infiltration of the neighbouring tissues, and no zone of hyperaemia around the benign neoplasm; but a microscopical examination of the removed growth should always be made. Fibroma and adenoma are very similar in aspect and consistence, but the former are much rarer than the latter, and grow more rapidly.

Prognosis.—The prognosis as regards life is always favourable; and the same may be said of the results of operative interference, as these tumours do not tend to recur after radical removal.

The *symptoms* manifested by all these benign growths are mainly due to mechanical interference with the action of the soft palate, with deglutition and phonation, or, if very large, with respiration; and the severity of the symptoms depends chiefly on the size of the growth. A papilloma on the tip of the uvula may give rise to the usual symptoms of elongated uvula. Fibromas are sometimes rather painful, especially if large; and, like any large growth in this region, may give rise to a sense of fulness and discomfort.

Treatment.—A papilloma should be cut off, and the tissues immediately around the seat of attachment included in the excised portion. An angioma may be removed by the galvano-cautery snare; but haemorrhage is apt to be considerable if precautions are not taken to prevent it. The other forms of growth need not be removed unless their presence occasion inconvenience or pain. Fibroma, especially of the rhino-pharynx, may give considerable trouble in removal. A description of the many surgical methods employed to overcome this difficulty is outside the scope of this work.

B. **Malignant Neoplasms.**—Both carcinoma and sarcoma occur with tolerable frequency in the fauces and pharynx.

The causes of malignant growths in this region are as obscure and ill-defined as are the causes of similar growths occurring in other parts of the body. Almost invariably the pharyngeal growth is primary, or due to extension from neighbouring structures; malignant disease in this region is very rarely secondary or metastatic.

The male sex is more frequently attacked than the female; especially is this the case with carcinomatous growths. It is rare for carcinoma to appear before the age of forty, and the great majority of all forms of malignant disease of the pharynx do not begin till after the fifth decade; sarcoma, however, may occur at any age.

Pathology.—The morbid anatomy of growths occurring in the pharynx does not differ in any way from the usual structure of similar growths in other regions. Primary carcinoma may occur in the soft palate, the pillars of the fauces, the tonsil, rhino-pharynx, or the lower pharynx at its junction with the oesophagus.

If arising in the soft palate it generally soon spreads to the tonsil, or from the pillars of the fauces to the tongue; whilst carcinoma of the lower pharynx tends to invade the larynx, so that at a later period it is often impossible to define the seat of origin. The growth presents an uneven surface and soon ulcerates. In its earlier stages epithelioma usually appears as a wart-like growth surrounded by hyperaemic, infiltrated tissue. At first it grows rather slowly, but, as it gets larger it rapidly spreads, invading surrounding structures in all directions. The growth sometimes forms a large tumour; but in other cases it soon begins to break down in the centre, the ulceration extending laterally as well as in depth, fresh nodules forming in the immediate neighbourhood of the hard, elevated margin, soon to be included in the ever-advancing ulceration. The base of the ulcer is covered with mucopus and breaking-down tissue, in the midst of which uneven ridges of the growth and ulcerating nodules are seen; but no granulation tissue is formed and scarring never takes place. The glands of the neck, and particularly those under the angle of the jaw, are soon extensively involved, whether the growth arise in the fauces or rhino-pharynx; when the laryngo-pharynx and oesophagus are the primary seat, the cervical glands are not so rapidly implicated.

The varieties of sarcoma met with in the fauces and pharynx comprise lymphosarcoma, round-celled sarcoma, mixed round and spindle-celled, spindle-celled, alveolar, melanotic, and myxo-sarcoma. Lymphosarcoma is probably the most common variety, and Mr. Butlin has suggested that a connexion probably exists between this form of growth in the fauces and lymphadenoma; and that in some cases the primary lymphoid deposit occurred in the faucial lymphatic tissues. Whilst it is beyond dispute that many cases of sarcoma of the fauces and tonsil display a mild malignancy, and that instances occur in which the faucial lymphoid tissue becomes involved in the course of lymphadenoma [*vide* Vol. IV. Part I. p. 465], it is most unusual to see the latter affection following sarcomatous growths in the fauces. Kundrat has observed lymphosarcoma in two cases of pseudo-leukaemia (lymphadenoma). Chiari states that leukaemia and pseudo-leukaemia are distinguished from lymphosarcoma of the throat by the infection of the lymphatic glands of the whole body as well as of the spleen and liver. Sarcoma grows somewhat rapidly; and when the growth has attained any size, the mucous membrane covering it is succulent and bright red in aspect, and it infiltrates and displaces the neighbouring structures. It begins in the tonsil, or in a lymphoid follicle of the mucous membrane of the soft palate, pillars of the fauces, or rhino-pharynx. It is less hard than epithelioma, and sometimes is soft and gives the sensation of a cyst or abscess. The rate of growth varies a good deal; in some cases it remains localised for a considerable period, or for a time may diminish in size. It spreads by extension to the neighbouring regions, and very generally invades the deeper tissues behind the angle of the jaw, so as to cause large swellings in the neck; sarcomas in the fauces or tonsil, on the contrary, not infrequently remain distinctly localised and more or less encapsuled for a long time, and then it is only when they extend beyond the limiting capsule that they increase rapidly and invade neighbouring structures and glands. Ulceration does not occur very early, and when it does it is usually superficial, and haemorrhage is very slight.

Lymphosarcoma of the throat is a rare affection, and generally occurs in middle-aged men. O. Chiari, who has made a special study of this variety of pharyngeal growth, states that it arises either on one of the tonsils or in the lymphoid follicles of the soft palate or rhino-pharynx; or the lymphatic glands of the throat, mostly on one side, may be attacked, and from them conical tumours may grow towards the throat, causing more or less narrowing of the space. Lymphosarcoma appears either as a definite tumour on the tonsil or as an infiltrating growth; in either case ulceration and breaking down of the surface soon occur. Thus, the larger tumours or the flat complex of smaller growths break down or suppurate. The resulting ulcers may heal partly or entirely, and even deep scars may be formed; yet soon fresh points of infiltration appear on the edge of the old ulcer, either as yellowish marrow-like nodules or simply as diffuse thickening. In this way the process spreads superficially. Sometimes the degeneration and the breaking-down process are accompanied by febrile disturbance. Like other forms of sarcoma, the lymphosarcomatous tumour may, temporarily, diminish in size, or even, for a time, apparently almost disappear.

Symptoms.—Carcinoma of the fauces is usually accompanied by pain of gradual onset, increased but not induced by deglutition, and lancinating in character, darting up to the ears. Salivation is often present. The voice becomes throaty or nasal in quality. A large growth may produce considerable dysphagia. Difficulty in swallowing, indeed, is often the earliest and most marked symptom in growths occupying the laryngo-pharynx; obstruction to nasal respiration and a sanious muco-purulent discharge occur rather when the rhino-pharynx is the seat of the disease. The breath becomes fetid when the tumour breaks down, and cachectic symptoms are seldom long delayed. In sarcoma and lymphosarcoma the symptoms generally consist chiefly of mechanical obstruction to respiration and deglutition, and alteration in the quality of the voice. In malignant disease of the naso-pharynx, when extending to the Eustachian tubes, the hearing, of course, also may become impaired. Pain is not usually a marked symptom, though generally noticeable when superficial ulceration has occurred. With the further progress of the growth deep ulceration with foul muco-purulent discharge, quickly-increasing extension of the disease, pain, loss of appetite and general weakness become manifest, and the patient then rapidly sinks.

Diagnosis.—The differential diagnosis of these forms of malignant growths often presents many difficulties: first, in distinguishing between the different varieties of malignant growths, a point of importance as regards prognosis and the advisability of operative interference; and, secondly, in distinguishing them from benign growths and various infective diseases.

Carcinoma differs from sarcoma in the early onset of pain, its irregular surface and infiltrating character, and the rapid secondary infection of the neighbouring glands; early fixation of the lower jaw and the early appearance of cachectic symptoms being in favour of carcinoma. The growths are usually harder on palpation, pale pink or even bluish-pink in colour, and are surrounded with a well-marked areola. Ulceration and haemorrhage occur early; the margin of the ulcer is ulcerated, raised, and irregular, and the floor is covered with characteristic cauliflower vegetations. A sarcoma is softer, smoother, and more succulent in aspect, covered with light pink or yellowish-pink mucous membrane, and ulceration is often delayed. The ulceration tends to remain superficial, but when the growth does break down there is a copious secretion of ichorous mucopus.

Lymphosarcoma differs from sarcoma and carcinoma in that these arise as more or less globular tumours, the ulceration of which shews less tendency to spread on the surface. Sometimes, however, they also appear in the form of a localised infiltration. The lymphosarcomatous ulcer is thickly covered with ichorous pus, but the floor does not shew cauliflower excrescences. Moreover, the lymphosarcomas are more apt to diminish in size; and the ulceration often heals, at any rate for a time.

In all forms a microscopical examination of removed fragments of the growth will generally decide the question of diagnosis; but this investigation must be carried out with great caution; first, we must obtain a piece which includes the deeper tissues of the tumour, for superficial portions may only shew normal or inflammatory tissue without evidence of malignancy; and secondly, we must distinguish between the varieties of carcinoma which closely resemble sarcoma.

The diseases most likely to be mistaken for malignant growths, and conversely, are chronic hypertrophy of the tonsils, tonsillitis, benign growths, syphilis, tuberculosis, lupus, diphtheria, infiltrations due to the invasion of pathogenetic organisms, such as the pneumococcus, and chronic retropharyngeal abscess. Chronic hypertrophy of the tonsils is in the great majority of cases bilateral, and is essentially a disease of early childhood and adolescence; and although enlarged tonsils dating from childhood may persist throughout life, yet an enlargement beginning in an adult, especially if unilateral, must always be regarded with grave suspicion.

A sarcoma may be mistaken for gumma, and especially for a gumma which looks yellowish and marrow-like just before breaking down; the only trustworthy distinction, indeed, consists in the failure of antisyphilitic medication. An ulcerating sarcoma, especially if ulceration be attended with febrile disturbance, may resemble acute tonsillitis or peritonsillitis; but the more gradual onset, slight constitutional disturbance, and absence of acute pain or tenderness, would serve to distinguish these affections, apart from the aspect of the growth. Sometimes an ulcerating sarcoma, and more especially a lymphosarcoma, is covered with a thick layer of mucopus which may simulate diphtheria or syphilitic ulceration. The presence of fresh nodules of growth around the margin, the shape of the ulcer, and the fact that it is always single, together with the general condition and freedom from albuminuria and from characteristic bacilli in a culture, would point to a malignant growth.

Syphilis is more likely to be diagnosed when a malignant growth has undergone extensive breaking down with deep ulceration. Very often only a microscopical examination of a removed fragment and the administration of iodide of potassium will settle the question. Yet it is always important to remember that a temporary subjective amelioration, and even a transient diminution in the size of a malignant growth, may be produced by the exhibition of iodide of potassium. Too much stress ought not to be laid on the history of a syphilitic affection, or evidence of old syphilitic scars and the like; for, on the one hand, in syphilis a history of infection often cannot be obtained, and on the other hand malignant disease not infrequently occurs in syphilitic subjects.

The peculiar apple-jelly-like nodules of infiltration around lupus ulcers should prevent an error in diagnosis as regards this disease.

Similarly ulcerating sarcoma may bear a strong resemblance to tuberculous ulceration; more especially is this true of lymphosarcoma. Tuberculous ulcers are more superficial, have mouse-nibbled edges, and are usually multiple. If no concomitant pulmonary symptoms are detected the examination of the muco-purulent secretion will reveal the tubercle bacilli; whilst the frequent pulse and nocturnal rise of temperature should lead to a suspicion of this disease. Of course the presence of pulmonary tuberculosis does not exclude the possibility of a malignant tumour in the pharynx.

A warty carcinomatous growth on the fauces may closely resemble a benign papilloma; yet it differs from it in growing from an indurated base, and in being surrounded by a zone of hyperaemia and infiltrated tissue. Similar points of distinction serve to differentiate malignant growths and fibroma; but a sarcoma may appear so truly benign in aspect, in rate of growth, and in the absence of any enlargement of neighbouring structures and glands, that the only means of diagnostic distinction may be in a microscopical examination of a removed fragment (the possible sources of error will be fully discussed in the article on malignant disease of the larynx, p. 249). The same difficulty may arise in the distinction of the rarer forms of benign growth, such as adenoma, from malignant growth. Microscopical and bacteriological investigation will differentiate malignant growths from infiltrations due to invasion of the tissues by the pneumococcus and other pathogenetic organisms.

Chronic retropharyngeal abscess occurs in an unusual site for sarcoma, and presents a smooth swelling which fluctuates on digital exploration, and is sometimes associated with cervical caries. Aspiration of the tumour would, of course, reveal its true character at once.

[TABLE

The following table, compiled by one of us (W. W.), summarises the main points of distinction between several of the diseases of the pharynx and fauces:—

CARCINOMA.	SARCOMA.	CHANCRE.
Symptoms. — Dysphagia is always an early symptom, and pain is considerable and persistent, but of gradual onset. Increased pain on swallowing becomes so great as to prevent the patient taking food. Saliva accumulates in the mouth. Early and well-marked cachexia, and rapid loss of flesh. *Physical Signs.*—Carcinoma always presents an enlargement with superficial irregularity of surface, which is light pink or bluish, and soon ulcerates with granular fissured surface, hard elevated margin, general cartilaginous hardness and fixedness. Ulceration not very depressed, covered with fetid mucopus. Early infiltration of neighbouring glands. Haemorrhage frequent and often profuse. sometimes fatal. Generally unilateral.	*Symptoms.*—Difficulty and pain in deglutition, sometimes very slight, and, until ulceration occurs, is chiefly mechanical. Saliva accumulates and dribbles from the mouth. Loss of flesh generally rapid. *Physical Signs.* — Sarcoma attains considerable dimensions before ulceration commences. The growth is red, fleshy-looking, and soft, surrounded by a well-marked bright red areola. Spreads to neighbouring regions and externally to the neck,—especially rapid is the extension of round-celled sarcoma. Haemorrhage is frequent and sometimes fatal. Generally unilateral.	*Functional Symptoms.*—The first symptom is a stinging pain in the tonsil, but with little pain on swallowing, which is never so difficult as in cancer or in tertiary syphilis. Cancer occurs in late middle life, but sarcoma may also occur in the young; chancre generally in young adults. *Physical Signs.* — The surface is very red, but there is always a well-defined erosion, with sharply-cut margin from the commencement. Induration or even stony hardness. The submaxillary glands early enlarged. Like cancer and tertiary syphilis, and unlike secondary, it is unilateral. No haemorrhage, only streaks of blood. No emaciation, early appearance of secondary rash, etc. Responds well to treatment.
SYPHILIS, Secondary and Tertiary.	TUBERCULOUS ULCERATION.	ACUTE TONSILLITIS.
Symptoms.—Often no pain whatever, and swallowing often difficult, never impossible. Wasting and cachexia in proportion to the difficulty in taking nourishment, and not very pronounced. No salivation. In secondary syphilis of the tonsils and fauces there is generally bilateral deposit of mucous patches and superficial ulceration, with well-marked purplish areola. In tertiary syphilis the tonsils are unilaterally affected by a deep perforating ulcer. The margins of the ulcer are often undermined, and overhang the deep-lying ulcer, the floor of which is covered with necrotic tissue. The sympathetic glandular enlargement is slight, and not painful as in cancer. Haemorrhage slight or absent. The rapid improvement under antisyphilitic remedies is always a valuable sign.	*Symptoms.* — Swallowing is usually very painful, and loss of flesh rapid, with nocturnal rise of temperature, and a general well-marked tuberculous cachexia is often present. There is early and rapid infiltration of the parts around, with very early tendency for fluids to return through the nose on swallowing. *Physical Signs.* — General pallor, with diffuse infiltration of the affected region. Early superficial, irregular, mouse-nibbled ulceration, with grey debris. In the earlier stages the deposits of miliary tubercles are very characteristic; these ulcerate and coalesce. No inflammatory areola. Haemorrhage generally absent. Usually concomitant disease of larynx and lungs.	*Functional Symptoms.* — Pain very marked from the commencement, great tenderness and difficulty in swallowing. Generally some rise in temperature. Usually both tonsils affected, though one after the other. *Physical Signs.*—Characteristic redness and inflammatory infiltration. Lacunar exudation, but no ulceration. May proceed to suppuration. *Chronic abscess* of the tonsil may be diagnosed by incision and discharge of pus. Responds well to treatment.

Prognosis.—The prognosis of carcinomatous growths in the pharynx is very grave. The only exception to this statement occurs in the case of small warty growths which appear on the soft palate or uvula, and which may be radically extirpated in an early stage.

A sarcoma occurring in the fauces is a slightly more hopeful affair, especially the less rapidly-growing spindle-celled variety and the lymphosarcoma. These growths may remain encapsuled for a long time, so that a relatively favourable prognosis is justified when secondary extension is slow to appear, inasmuch as a radical operation may be completely successful. It is hardly necessary to say that any form of malignant growth, especially in a region so rich in lymphatic supply and so difficult of access as the tonsils or fauces, not to mention the rhino-pharynx, is peculiarly grave; but on the other hand there is too great a tendency to overlook the fact that cases have been recorded in which a radical operation has been completely successful, especially of late years. While fully realising the importance of not regarding malignant disease in this region as invariably hopeless, a lasting freedom from recurrence after a radical operation is unfortunately the exception.

Treatment.—From a therapeutic standpoint the importance of early diagnosis of malignant growths cannot be too strongly emphasised. In considering the advisability of attempting a radical operation, the surgeon will be guided not only by the situation and limitations of the growth, but also by the particular variety of malignant tumour to be dealt with; for an encapsuled or well-defined sarcoma, especially if it be a lymphosarcoma or a spindle-celled sarcoma might be permanently removed, whereas a similar procedure for encephaloid cancer would almost certainly be unsuccessful. We are reluctantly bound to say that later experience has not justified the more hopeful views expressed in the first edition.

The choice lies between removal through the mouth by the knife, snare, or ecraseur, and lateral pharyngotomy. It is impossible, however, to lay down rules for guidance in every case; each must be judged on its own conditions.

On the other hand, growths may require partial removal when there is danger of suffocation or difficulty in swallowing; and pharyngeal or oesophageal constriction may necessitate gastrostomy or lateral oesophagostomy.

When a radical operation is impossible, or has been followed by recurrence of the growth, all that can be done is to maintain as long as possible the patient's strength by nourishing diet and suitable tonics, and to alleviate pain by opium. Ulcerating growths should be cleansed with antiseptic sprays and gargles.

In lymphosarcoma, as in lymphadenoma, arsenic in the form of Fowler's solution in steadily increasing doses is not rarely followed by great improvement and even by apparent disappearance of the new growth. Unfortunately, however, this improvement is almost always only temporary and followed by reappearance of the neoplasm, whilst the drug generally seems to lose its effect. It is well, therefore, not to be too optimistic in one's prognosis, should an almost miraculous effect at first follow the administration of arsenic. Trypsin might be tried in inoperable cases. Dr. Beard reports his investigations on mice inoculated with Jensen's cancer, and shews that the trypsin appears to cause the disappearance of the neoplasms. One of us (W. W.) has tried trypsin injections, 10 to 30 c.c., on alternate days, in a case of inoperable epithelioma of the palate and faucial pillars, but without any appreciable effect.

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For Diseases and Tumours of the Palate, see also Vol. III. pp. 315-317.

PHARYNGEAL NEUROSES.—(*a*) **Motor Neuroses.**—The motor neuroses of the pharynx may be conveniently divided into spasmodic neuroses and paralyses.

Spasm of the pharyngeal muscles is a rare affection, and is generally met with in nervous and hysterical patients. It may occur in association with various acute inflammatory processes, such as acute tonsillitis. It is a prominent symptom in hydrophobia, and has been observed in cases of cerebral tumour. Courmont records a case of tonic spasm in tabes. Clonic spasm of the levator palati ("nystagmus of the palate") gives rise to a peculiar clicking sound audible to the patient and those around. The cause is obscure. By some observers the affection is regarded as a reflex neurosis. In a number of cases this clonic spasm is met with in organic diseases of the brain, particularly, as some recent observations have shewn, in affections of the mid-brain, and it may occur either as an isolated phenomenon, or be associated with similar rhythmical clonic contractions of the tongue and the larynx, and with nystagmus of the eyes.

In some cases local application of the galvanic current has proved useful in relieving spasm. When due to hysteria or associated with neurasthenic constitutional states, nervine tonics, rest, and change of air are indicated.

Paralysis.—The experimental results of Sir V. Horsley and Dr. Beevor shew that the soft palate and uvula, the levator palati, and the pharyngeal constrictors are innervated by the spinal accessory fibres in the pharyngeal plexus, and not by the vagus. The tensor palati is supplied by the fifth nerve. Thus, paralysis of the soft palate may be caused by central nerve lesions implicating the spinal accessory, or by peripheral neuritis and pressure on the nerves to this region, or the paralysis may be myopathic.

The paralysis is generally unilateral, but may be bilateral.

Paralysis of the palate from bulbar disease may be due to acute or chronic myelitis implicating the spinal accessory nuclei, to bulbar apoplexy or embolism, to tumours, or to basic meningitis.

Acute bulbar paralysis is characterised by the sudden onset of giddiness, headache, and sometimes vomiting, with unsteadiness of gait, but with no loss of consciousness. The voice becomes nasal and thick, and, the lips and tongue being involved, articulation is difficult. The dysphagia increases, and finally respiration becomes irregular, and the pulse small and frequent from the progressive implication of the various bulbar nuclei.

Chronic bulbar or glosso-labio-laryngeal paralysis generally begins in the tongue; then the lips, velum palati, and pharyngeal constrictors are affected; and often the abductors and internal tensors of the vocal cords likewise. Speech becomes nasal in tone, articulation very imperfect, and swallowing very difficult and liable to result in the food passing into the larynx. The vago-accessory nuclei being concerned, the pulse is often persistently frequent, respiration may become shallow and irregular, and attacks of periodic dyspnoea are not uncommon towards the end. Unilateral paralysis of the tongue, palate, and larynx, first described by Dr. Hughlings Jackson, and in a number of cases associated with paralysis of the trapezius and sterno-mastoid, may be of central or of peripheral origin. The understanding of this triad of symptoms, which seemed simple enough when it was thought that the motor innervation of both the palate and the larynx was supplied by the spinal accessory, has become much more difficult, since the weight of evidence goes to shew that the motor innervation of the larynx is supplied by the vagus.

Post-diphtheritic neuritis is the most common cause of palatal paralysis. A similar condition may probably be caused by membranous sore throat not associated with the Klebs-Löffler bacillus, and from acute lacunar tonsillitis (*vide* p. 166).

Paresis of the palate and constrictors of the pharynx may be due to hysteria, or to general weakness in the anaemic and debilitated.

The *symptoms and signs* of paralysis vary as it is unilateral or bilateral. When the lesion is unilateral, the uvula is drawn towards the healthy side, and the velum palati is drawn down by the palato-pharyngeus and palato-glossus on that side; if bilateral, the velum palati hangs loosely and does not respond to local stimulation, the voice is nasal, and fluids escape by the nose during deglutition. As the paralytic condition of the pharyngeal constrictors increases, deglutition becomes more and more difficult, and the difficulty in swallowing fluids is always greater than for solids, in contradistinction to the difficulty in swallowing due to obstruction, when, as we should expect, it is first noticed, and is always more pronounced, as regards solids.

It is necessary to distinguish from true pharyngeal paralysis the very similar appearances which may result from inflammatory exudation and mechanical interference with the movements of the soft palate due to syphilis and other forms of local disease.

The view that paralysis of the palate is due to and accompanies paralysis of the facial nerve is practically no longer accepted.

The *treatment* of pharyngeal paralysis will, of course, depend on the causes. In many cases local treatment is obviously of no use whatever. It is, however, remarkable that faradisation of the palate and pharynx in chronic bulbar paralysis, as recommended by Erb, appears to retard—for a time at any rate—the progress of the difficulty in deglutition. Post-diphtheritic paralysis should be treated by hypodermic injections of strychnine and local faradisation.

(*b*) **Sensory Neuroses.**—*Anaesthesia*, partial or complete, may be unilateral or bilateral. The commonest cause is diphtheria; but it may occur in hysteria, bulbar paralysis, and in insanity; it is also produced by pressure on the glosso-pharyngeal nerve by tumours near the exit of the nerve from the skull, or by intracranial tumours, gummas, etc. It is nearly always associated with neuroses of sensation and paralyses of the velum and larynx.

Hyperaesthesia and paraesthesia of the pharynx are often met with, apart from any organic disease, in anaemic and neurotic patients; but in many cases some slight affection of the tonsils or granular pharyngitis is the source of a discomfort altogether out of proportion to the cause. Very similar painful or uncomfortable sensations in the pharynx are often found in gouty patients, in the early stages of pulmonary phthisis, of cancer of the pharynx, and so forth. Both in men and women there is a very intimate connexion between the whole region of the upper respiratory tract, the nose, pharynx, and larynx, and the sexual organs; and many of the more obscure neuroses of these regions have a sexual basis. That such a special if somewhat mysterious connexion exists, has long been known. Its physiological correlation in man is found in the sudden development of the larynx during the time of puberty, particularly in males, accompanied by characteristic changes in the voice, known as the "break of the voice"; whilst in women a slight huskiness of the voice and other indefinite phenomena are often noticeable at the time of menstruation; practised singers often notice a deteriorating influence of the menstrual period on the voice. The effect of castration of boys in modifying these changes in the larynx is well known. In the lower animals this physiological connexion is well seen, again, in the "roaring" of the otherwise silent stag at the rutting time and in the vocal auto-intoxication in which the capercailzie cock indulges during the corresponding period.

Whilst the influence of the sexual organs on the respiratory organs is most obviously recognised in the larynx by the alterations in the voice, many of the purely subjective neuroses are referred indefinitely to the throat region generally. As Schadewaldt pointed out, the power of localising sensations felt in the throat is very defective physiologically as well as pathologically; "the sensations in the most different parts of the organs of the neck are, as a rule, jointly referred to a region in which, so to speak, the joint sphere of sensation (the sensorium commune, according to analogy) of the entire throat is situated." This region is the front part of the neck, the "laryngo-tracheal region" (Gottstein). It is therefore of no use to attempt to distinguish the subjective sensory neuroses of the pharynx from the rest of the upper respiratory tract, as an attempt of this kind might easily lead to therapeutic mistakes.

Chlorotic and anaemic girls, and women at the climacteric period, very frequently suffer from paraesthesia of the throat region; but it is the latter class who afford the great majority of instances and in the aggravated forms. The "sensory throat neuroses of the climacteric period," as they have been called by one of us (F. S.), may be classed under two headings, "paraesthesia" and "neuralgia" cases, the former class being the more frequent. We have never seen a case of anaesthesia of the throat due to the climacteric period. The majority of cases of climacteric throat neuroses occur in women who are by no means of a neurotic or hysterical type. Most frequently patients complain of unpleasant sensations which often enough cannot be described exactly; in some cases they are general, in others they shift from one part to the other. In other cases, again, the patients speak of general or partial "soreness," "dryness," "tickling," of a desire to be constantly "scraping," or "hawking" and "hemming," of sensations of "choking" or "strangulation," or of a feeling as if the throat were "wooden"; very frequently there is a sensation as of a foreign body, variously compared to a crumb of bread, a bone, a hair, or a needle, or a constant desire to "swallow empty," or feelings of heat or cold. But in very many cases one sees how the patients strive in vain to describe exactly what they feel, and to define the seat of the sensation. Much less frequently there are "neuralgic" sensations, described as a fixed pain on one side of the throat, sometimes radiating to the ear and temporarily diminished by swallowing.

The intensity of these neuroses varies most remarkably; in some, the sensations are merely felt as an inconvenience; in others, the subjective troubles are of a more severe kind. The patients not rarely even cry while relating the history of their ailment; and the general depression accompanying the affection is sometimes so great that in a good many cases the patient dreads cancer, consumption, syphilis, or some other organic disease of the throat.

The throat symptoms complained of may be the only sign of the approaching change of life; or sometimes may even precede the menstrual irregularities; in other cases they follow the usual uterine disturbances of the climacteric period, or are associated with dyspepsia, insomnia, and other complications commonly observed at the menopause.

Objectively there may be little or absolutely nothing to be seen in the throat. In some cases a few small pharyngeal granulations, or a slight enlargement of the lingual tonsil, or some hardly noticeable thickening of the lateral folds of the pharynx, are detected. It is important to guard against two sources of error: namely, of overlooking some actual and tangible cause of the affection, or, on the other hand, of wrongfully attributing the neuroses to any slight abnormality. The objective symptoms in the cases which belong to the domain of paraesthesia and hyperaesthesia, and of the sensory neuroses of the climacteric period, are either conspicuous by their absence, or the changes found are so slight as to make it extremely unlikely that they can be held responsible for the subjective phenomena. On the other hand, it ought to be remembered that paraesthesia, hyperaesthesia, or neuralgia of the throat may be the first sign of malignant disease of the part or of its neighbourhood; and that the age at which the climacteric neuroses come under observation is identical with that in which the beginning of malignant mischief is most frequently observed. Before arriving at a diagnosis of sensory throat neuroses, we must first, by careful examination, exclude chronic pharyngeal catarrh in its definite forms, considerable nasal stenosis, a foreign body, considerable enlargement of the lymphatic tissue at the base of the tongue, general anaemia of the pharynx—particularly in cases of commencing tuberculous disease of the lungs, and general neurasthenia or hypochondriasis.

The *treatment* of sensory neuroses will of course depend on the cause. In bulbar paralysis and other central organic nervous affections treatment is practically useless; whilst in post-diphtheritic anaesthesia the treatment is the same as that indicated in the motor paralysis with which it is almost invariably associated; namely, gentle faradism or galvanism combined with hypodermic injections of strychnine. As regards hyperaesthesia and anaesthesia, the treatment must be directed to improve the general health and to remove any possible local cause. In cases of neuralgia particularly the probe ought to be used in order to ascertain whether there be any tender spot in the painful part; anaemia and chlorosis should be treated by the cautious use of iron and arsenic. In cases in which there is any reasonable doubt whether the neuroses are due to the climacteric period or to some local mischief, the latter should, of course, be treated, but in these cases much judgment is necessary, and we would particularly warn the practitioner against regarding every small apparent departure from the normal as responsible for the occurrence of these climacteric neuroses. In such cases the patient should be given a simple explanation of the state of matters and should be encouraged to look forward with confidence to its spontaneous disappearance in course of time. The usual local remedies—astringents and caustics—are generally quite useless, or have but a very transitory effect; and their indiscriminate use is to be strongly condemned, inasmuch as this whole period of a woman's life is in itself associated often enough with a state of mental depression: thus, after the failure of local means, patients are prone to become still more depressed and more than ever convinced that their ailment is really of a serious nature. This caution applies particularly to the use of narcotics, such as opium, cocaine, bromide of potassium, and the like, which, whether locally or constitutionally employed, after a very short time lose their effects, and the patients either become enslaved by a pernicious habit, or by abstinence from the accustomed drugs their general and local sufferings are considerably increased. In severe cases of paraesthesia, and more particularly in cases of neuralgia, we should use those drugs only which cannot do any possible harm, such as menthol in spray and general tonics. The best effects are certainly obtained in those climacteric cases in which the throat neuroses are associated with considerable increase in bulk, digestive disturbances, and gouty manifestations. In such cases, if the patients can be persuaded to go through a mild course of the mineral waters of Carlsbad, Marienbad, Kissingen, Aix-les-Bains, or Vichy, a disappearance of all the symptoms complained of and a restoration of balance are often much sooner obtained than in ordinary forms of climacteric neurosis. But in the great majority of cases no treatment other than moral influence is either necessary or desirable.

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Diseases of the Uvula

The uvula being practically a part of the soft palate, it is very frequently implicated in diseases affecting that region, whilst its affections present some special features.

Congenital absence of the uvula occurs especially in association with cleft palate; or the uvula may be more or less completely bifid, representing an incomplete cleft palate.

Inflammatory Affections.—In acute inflammatory diseases of the pharynx, from whatever cause, the uvula generally becomes inflamed; and in septic inflammations it is especially liable to become so enormously swollen and elongated that it may even approach the size of the little finger. Very rarely it is long enough to be grasped between the teeth when coughed forward to the front of the mouth, or to fall into the larynx when drawn backwards and downwards.

An oedematous uvula may be freely scarified, and, when the inflammation is acute, sucking of ice may be grateful to the patient. In other respects the treatment does not differ from that of the pharyngeal affection with which it is associated.

Chronic uvulitis is usually associated with chronic pharyngitis, the velum palati and uvula being relaxed and congested, and the latter frequently elongated; whilst enlarged venules and mucous glands are found scattered over the surface.

Elongated Uvula.—An exaggerated importance is only too frequently attached to the uvula as a source of many and various symptoms in the region of the throat; we must therefore at once express our decided opinion that it is in very rare cases only that the condition of the uvula can properly be regarded as the cause of any notable symptoms; in the vast majority of patients the symptoms which are sometimes attributed to the uvula are really due to morbid conditions in other parts of the upper respiratory tract.

We may conveniently classify cases of elongated uvula into two subdivisions, viz. (*a*) those in which the uvula is merely relaxed, the mucous membrane extending some distance below the muscular structures but without congestion or hypertrophy; and (*b*) those in which hypertrophy and chronic congestion are present, often associated with degeneration of the glandular structures of the naso-pharyngeal mucous membrane.

Symptoms.—In a great many cases, unless the elongation be very well-marked, there are no symptoms whatever. In the milder cases, in which there is merely relaxation of the soft palate and uvula without hypertrophy or congestion, the symptoms complained of are mainly impairment of the quality and strength of the voice, and are mostly observed in professional singers. But the alteration and impairment of voice are often due rather to the relaxation of the soft palate, interfering with the proper movements of the uvula in singing high notes, than to the elongation of the uvula in itself. In well-marked cases patients usually complain of continual hawking, with a sense of some foreign body in the throat. The cough is sometimes very severe and persistent, particularly on lying down at night. The constant titillation at the back of the tongue not infrequently results in vomiting; this is especially in the morning or after meals, and, if the elongation be so considerable that the uvula reaches down to the larynx, laryngeal spasms may occur. In men much addicted to abuse of tobacco and alcohol the last-named symptoms are particularly frequent. In a few and very rare cases the constant pain and irritation in the throat, persistent cough, and frequent vomiting, may result in emaciation and weakness; whilst the recurrent haemorrhage from rupture of enlarged vessels in the pharynx may, in conjunction with the other symptoms, give rise to the suspicion of serious lung mischief.

Treatment.—When really necessary, and when all other sources of the symptoms presented have been excluded, ablation of the uvula should be performed; but here again we would emphatically state that in our opinion the operation is very rarely necessary.

The cases in which uvulotomy are required are—(i.) In professional singers suffering from loss of vocal tone without appreciable affection of the larynx, in whom the uvula is elongated, thickened, and relaxed; (ii.) in cases in which the elongation is so considerable that it becomes sucked into the larynx and produces attacks of suffocation, especially during sleep; (iii.) when a long and thickened uvula is associated with a persistent tickling cough, and when, after careful examination of the pharynx and larynx, all other possible causes for the symptoms have been excluded; (iv.) in malignant disease of the uvula; (v.) and, finally, in cases in which a much-elongated uvula is an obstacle to the performance of delicate intra-laryngeal operations. When cases are properly and judiciously selected the result is most gratifying, sometimes altogether out of proportion to the relatively trivial operation. The great amount of benefit that may be derived from such a simple procedure as removal of the uvula was well illustrated in a case observed by one of us (W. W.). The patient had in fact been treated for supposed pulmonary tuberculosis. He was certainly very feeble and emaciated, but after his uvula was partly removed the improvement and final recovery were rapid: three pounds in weight were gained during the first fortnight.

In performing uvulotomy, the parts having been well cocainised, the tip of the uvula—unless the uvulotome be used—should be seized with forceps and gently drawn forward. The redundant portion is then removed by one cut with a pair of curved blunt-pointed scissors. By operating in this manner the cut surface comes to be posterior, and irritation by food on deglutition is avoided. The whole uvula should not be removed, but the redundant part only. If too much has been taken away, patients often complain of "want of power" in the throat, and sometimes of difficulty in speaking or reading aloud.

For a few days after the operation the patient should avoid talking, and the food should be soft, bland, and cold. A spray may be used containing cocaine and phenazonum dissolved in glycerin and water; or a mild morphine and cocaine pastille may be sucked at intervals, especially before meals, or, perhaps best of all, anaesthesine or orthoform may be insufflated. The pain and irritation resulting from the operation are occasionally considerable, and last from two to five days, being sometimes disproportionate to the smallness of the cut surface. Secondary haemorrhage may occur two or three days after the operation, hence the importance of the patient avoiding all hard or even solid food.

Chronic Infective Diseases.—Syphilis or tuberculosis, for instance, may attack the uvula, the symptoms and treatment being the same as in these diseases when affecting the fauces.

Growths of the uvula comprise papilloma, mucous polypus, and carcinoma. (See "New Growths of the Pharynx," p. 146.)

Paralysis of the uvula occurs in association with paralysis of the velum palati. Paralysis of the uvula alone may occur as a consequence of diphtheria.

DISEASES PECULIAR TO THE TONSILS

Introductory Remarks.—We have no certain knowledge of the physiological functions of the faucial, lingual, and pharyngeal tonsils; but Philip Stoehr has drawn attention to the fact that their epithelial covering shews gaps large enough to allow the passage of leucocytes; an enormous transit of such cells undoubtedly occurs into the tonsils without actual destruction of the epithelial strands. The leucocytes or phagocytes are protective against the invasion of the pathogenetic microbes which are brought into the fauces and naso-pharynx by inspiration; although it may well be that they have other unrecognised functions also to fulfil. However this may be, whilst the fissures and crypts of the tonsil form convenient resting-places or "traps" for microbes, the peculiar anatomical arrangement of their epithelial covering opens the gates to their invasion; and thus it is easy to understand how the tonsils, especially if the vitality and resisting power of the tissues be flagging, may form a portal for the invasion of the system by pathogenetic organisms. Recent researches furnish abundant proof of the correctness of these surmises. In addition to the demonstration of tubercle and other bacilli in the tonsils by Buschke, Schlenker, Krueckmann, Strassmann and Dmochowski, Dieulafoy, and Cornil, the investigations of Prof. Sims Woodhead, Krueckmann, and others prove that the bacilli do get through the epithelium of the throat, and into the cervical lymphatics in tuberculous patients, and in animals fed on tuberculous food. Jonathan Wright has demonstrated numerous tubercles with many giant-cells and a few tubercle bacilli in a piece of naso-pharyngeal adenoid tissue removed by Chappell from a patient exposed to tuberculous infection, in whom clinical evidence of pharyngeal tuberculosis ensued after operation for adenoids. Park inoculated guinea-pigs with fragments from the same piece with positive results. The possibility of direct and primary tuberculous infection of the tonsils, or of the naso-pharyngeal tonsil, is therefore beyond doubt; but that this is relatively rare is probable. Thus, Wright examined histologically the faucial tonsils removed in 5 cases and adenoids from 7 cases, fragments from each being introduced into the peritoneal cavities and subcutaneous tissues of guinea-pigs, and all, without exception, with negative results. He therefore agrees with Hodenpyl, who examined 200 sections of pharyngeal lymphoid tissue for tubercle with entirely negative results, that tubercle bacilli are rarely present in the tonsils or in the adenoids. As the result of an investigation into the part of the tonsil in scarlatinal infection, Dr. Walter Dowson was led to the conclusion that the tonsillar lesion and cervical bubo of scarlet fever are the analogues of the chancre and bubo of syphilis. That diphtheria preferentially makes its first appearance on the tonsils is well known, and in a series of cases of septic inflammation of the throat and neck published by one of us (F. S.), acute tonsillitis formed one of the initial symptoms in a considerable proportion; and the researches of Sendziak and others have proved that acute lacunar tonsillitis is due to direct infection by streptococci, staphylococci, and pseudo-diphtheria bacilli. The relation of tonsillitis to rheumatic fever was observed by Trousseau, who, writing in 1865, described the rheumatic sore throat, and went on to say that "these phenomena . . . are fugacious, like most affections of a rheumatic nature. The next day . . . another pain will occupy the neck . . .; whilst the day after, one of the shoulders may be the part attacked. . . . Patients who have had several attacks of this kind are able at the outset to distinguish the rheumatic affection of the throat." Lennox Browne and Morell Mackenzie also emphasised this relationship. Finally, Suchannek and also Goodall have summarised the previous observations on the connexion of rheumatism with tonsillar affections, and bring forward evidence to support the opinion we have long held, viz. that it is highly probable that in many cases the virus of rheumatic fever also obtains its entrance into the organism through the tonsils.

In view of such facts as these, it is obvious that the tonsils play a very much more important part in admitting the various infecting microbes than has hitherto been conceded; and we have no doubt that their condition merits close attention when the question of the etiology of infectious diseases is discussed.

Cartilaginous nodules and *bony trabeculae* are not very rare in the tonsils. Dr. H. Walsham and others suggest that they are of fetal origin, that is—they are cartilaginous nodules derived from the second branchial arch; on the other hand they may be metaplastic as maintained by Kanthack, the fibrous tissue becoming changed into cartilage or bone. They are, however, of no clinical import except that they occasionally cause some trouble in the course of tonsillotomy.

Acute Tonsillitis.—We distinguish three clinical forms: (i.) *Superficial or lacunar tonsillitis*, with diffuse inflammation of the mucous membrane of the tonsil and accumulation in the crypts of a great number of bacteria (small diplococci especially), and of lymphoid corpuscles contained in a fibrinous network and appearing in the mouths of the distended crypts as discrete patches of yellowish exudation. While this exudation is mainly on the surface of the epithelium, small necrotic points have been observed where the process has extended into the superficial layers of tissue (Sokolowski and Dmochowski). (ii.) *Parenchymatous tonsillitis*, in which the deeper tissues of the body of the tonsil are mainly inflamed, the amount of swelling being considerable. (iii.) *Peritonsillitis*, in which the connective tissues in front of and above the tonsil are chiefly implicated. Probably in most cases it is really an inflammatory affection of the extensions of the supratonsillar fossa. Suppuration is especially prone to follow peritonsillitis, but lacunar and parenchymatous tonsillitis may also end in suppuration.

Etiology. — Acute lacunar tonsillitis is undoubtedly an infectious disease, which is associated with various micro-organisms, and may be induced by a number of causes. It is usually believed to be especially prevalent in the late autumn and early spring, but observations continued by one of us (F. S.) for several years at St. Thomas's Hospital, and by the other (W. W.) at the Bristol Royal Infirmary, have shewn that the greatest number of cases applying for treatment of lacunar tonsillitis occurred in the middle of the summer. There have been numerous instances in which the affection has run through a household, attacking its various members in turn. B. Fraenkel and Macintyre drew special attention to the infectivity of acute tonsillitis when introducing a discussion on this point in 1895. Overwork, anxiety, and all causes, whether local or general, which lower the resisting power of the tissues render the individual more liable to infection. Thus, chronic hypertrophy and degeneration of the tonsils indirectly dispose to attacks. In many cases attacks of arthritic rheumatism directly precede or follow the tonsillitis; and, indeed, the causes of rheumatism, such as exposure to cold and damp, or to sudden changes in temperature, are likewise important causes of tonsillitis. Tonsillitis is commonly one of the initial symptoms in measles and scarlet fever, and is often met with in diphtheria and secondary syphilis. In not a few cases tonsillitis is due to septic poisoning; and the frequent occurrence of attacks of tonsillitis in a household, like all forms of recurrent sore throat, should lead us to suspect bad drainage. Again, it may be due to injury, as by a spicule of bone in the food, or by mechanical injury; and it is sometimes set up by the presence of calcareous cheesy masses in the crypts.

Tonsillitis is essentially a disease of early adolescence; but may occur at any time of life from earliest infancy to extreme old age.

The part usually first affected appears to be that corresponding to the posterior boundary of the supratonsillar fossa, and Dr. Paterson has explained this partly by the shutting off of the fossa, owing to the extremely narrow opening left by the upper part of the plica tonsillaris, which causes retention of invading organisms, etc., and partly by the distribution of lymphoid tissue which is disposed in a very loose, open network, whilst the crypts and lacunae here are large and open freely, communicating with each other.

The symptoms vary very much in degree in different cases. The attack generally begins with soreness and stiffness in the throat for one day, with aching in the back and limbs, headache, and general feeling of malaise, followed by a rigor with sudden rise of temperature which soon reaches 104° to 105° F.; the pulse is frequent, full, and bounding. With the onset of swelling and inflammation of the tonsils, pain darting up to the ears, and dysphagia, are prominent symptoms, and are often agonising. The constant desire to swallow is dreaded because of the pain of it; the accumulating saliva therefore dribbles from the mouth. The tongue becomes thickly coated, and the bowels constipated. The urine is scanty, high coloured, rich in urea and urates, and sometimes contains albumin. The spleen is often enlarged. The mouth can scarcely be opened, partly on account of the swelling of the tonsils, and often of the submaxillary tenderness and tumefaction also. Catarrhal inflammation always extends more or less from the tonsils to the fauces and pharynx. The rhino-pharyngeal tonsil is likewise involved with much greater frequency than is generally believed; and this must very often be held to account for the deafness and tinnitus due to stoppage of the Eustachian tubes. The lingual tonsil is also liable to attack; Sendziak observed this complication in twelve patients out of 133 cases of lacunar tonsillitis. When suppuration has begun, the pain and tenderness are greatly increased. In suppurative peritonsillitis, though the pain is more pronounced than in the first two clinical varieties, the general disturbance and febrile symptoms are often slighter. In the lacunar and parenchymatous forms both tonsils generally become affected, though as a rule one tonsil is affected earlier or to a greater degree than the other: peritonsillitis is almost always unilateral.

The course of the affection is rapid, seldom lasting more than from two days to a week, and ending in resolution or suppuration; but the subsequent prostration may be extreme.

Diagnosis.—In peritonsillitis the tonsil itself is often slightly inflamed or not at all—a redness and smooth bulging may be observed on one side of the soft palate; in parenchymatous and lacunar tonsillitis the tonsils themselves are always red and swollen. In the later form the discrete patches of yellowish exudation from the crypts are ordinarily characteristic enough to prevent confusion with diphtheria; but in not a few cases a differential diagnosis is impossible without resorting to bacterial cultures, for the lacunar exudations may spread beyond the crypts, and, becoming confluent, may form a sort of false membrane sometimes adherent to the tissues and indistinguishable from a diphtheritic membrane. The points in favour of diphtheria are (*a*) a false membrane of a greyish-white colour, thick, and firmly adherent, and involving the pillars of the fauces, the soft palate, or uvula; (*b*) the early presence of albumin in the urine in considerable amount, with a low or only slightly raised temperature, little pain, and unilateral affection. Submaxillary swelling and enlarged cervical lymphatic glands are common to diphtheria and tonsillitis.

We must further remember that, instead of the usual tough, grey, adherent false membrane, diphtheria may be associated with a soft, pultaceous exudation which may be restricted to the crypts, or may occur without any visible false membrane. In these doubtful cases it will always be advisable to leave the diagnosis in suspense for twenty-four hours until cultures have been made.

Prognosis. — In simple tonsillitis the prognosis is nearly always favourable; but we must be on our guard lest we overlook the earlier manifestation of the more virulent septic forms, which may result in oedematous, erysipelatous, or phlegmonous laryngitis, or in purulent cervical cellulitis (angina Ludovici), spreading to the mediastinal glands; or in general infection with resulting endocarditis or pericarditis, infective phlebitis, orchitis, or ovaritis. Further, tonsillitis may be the precursor of an attack of acute rheumatism, or less frequently of acute gout. Instances of cardiac complications arising in the course of acute tonsillitis, even without any arthritic symptoms of rheumatism, have been observed by Packard.

Very rarely paralytic sequels have occurred; and though no doubt palsies of the soft palate, ocular muscles, or other parts are strong presumptive evidence of the diphtheritic nature of the case, yet in a few of these careful investigation has failed to reveal the Klebs-Löffler bacillus. Cases of death from suffocation in young children by excessively swollen tonsils are recorded; and it has been necessary to perform tracheotomy to prevent asphyxia from laryngitis consequent on the tonsillitis. Death has occurred from rupture of a tonsillar abscess and escape of pus into the larynx.

Treatment.—From the outset the bowels should be kept freely moved, preferably by saline aperients. If the temperature be much above the normal, six grains of sulphate of quinine should be given every four hours till it is reduced. In rheumatic cases, in which there is much aching pain in the limbs and back, tincture of guaiacum, or fifteen to twenty grains of salicylic acid, or the sodium salt, or aspirin, given every two hours, will often alleviate the symptoms. Tincture of aconite in small and frequently repeated doses is useful in young children. In parenchymatous tonsillitis especially, guaiacum lozenges should be prescribed, six to eight being slowly dissolved in the mouth in the twenty-four hours. Formamint lozenges also are often useful. Spraying or painting the tonsils with 8 to 10 per cent solution of protargol or argyrol seems to check the tonsillar inflammation and affords considerable relief. Gargling with dilute solutions of chlorate or permanganate of potassium to which phenazonum, ten or fifteen grains to the ounce, has been added, is most useful; and sucking ice often gives considerable relief. But if the pain and swelling are pronounced, gargling may be out of the question; then hot fomentations applied to the neck and lower angle of the jaw, or a spray of cocaine (2 to 5 per cent) or of menthol (10 to 15 per cent) dissolved in colourless oil of vaseline or in oleum adepsin., will lessen the pain. The two solutions may be combined; for menthol has the additional advantage of being antiseptic. Firm compression with the tips of the fingers applied just in front of the external auditory meatus will greatly relieve the pain on swallowing. Any indication of suppuration should be watched for, especially in peritonsillitis; in such cases the inhalation of steam or gargling with warm water relieves the pain and tends to make the pus point. In cases of peritonsillitis, when the soft palate is seen to be bulging forwards and fluctuation is felt through it, the incision always ought to be made—not, as is still sometimes done, behind the palate into the substance of the tonsil itself, but through the palate in the direction from without and below, inwards and upwards.

The tonsils, as a rule, should not be removed while inflamed; to this rule, however, two exceptions may be given; namely, when in children respiration is greatly embarrassed by the tonsillar swelling, and when in adults tonsillitis has repeatedly occurred, but removal during the period of quiescence is for one reason or another impossible.

Patients are generally much weakened by tonsillitis, and need feeding up and suitable tonics, such as iron and quinine.

As tonsillitis, or at any rate the acute lacunar form of it, is certainly infectious, it is well to advise the patient's friends to avoid such immediate contact as kissing; children and persons specially prone to the affection should keep away, but strict isolation is not so necessary as in the case of diphtheria.

Acute Ulcerative Tonsillitis.—Occasionally superficial ulceration of the tonsils occurs, probably analogous to the simple ulcer of the pharynx, the so-called angina ulcerosa benigna. In other cases the ulcers are multiple, circular or oval, deep, with clean-cut margin, non-indurated base, floor sanious or covered with greyish-white exudate, and without marked inflammation of the surrounding area. This is acute ulcerative lacunar tonsillitis described by Moure, and must be distinguished from the mild septic ulcerations such as those occurring in medical students exposed to septic conditions in the dissecting-room or dead-house that German observers have called angina nosocomii. The benign ulcers appear when health and strength are impaired, in anaemic persons especially when reduced by fatigue, influenza, etc., and particularly in the subjects of rheumatism. The ulcers develop on one or more crypts, and are not apt to become confluent.

The *symptoms* are those of a mild lacunar tonsillitis, and though sometimes running a slow course the process is always benign; there is no fever, and no notable general disturbance.

Deep ulceration sometimes follows rapidly the phlegmonous inflammatory swelling in acute septic tonsillitis. But the inflammation and ulceration may be almost entirely confined to one or both tonsils. One of us (W. W.) had a case in which the tonsil sloughed away bodily in three or four days. The bacteriological examination shewed the presence of one of the forms of putrefactive organisms found in sewage, and the patient undoubtedly contracted the complaint from sewage contamination.

Chronic Enlargement of the Tonsil.—*Causes.*—Hypertrophy of the tonsils is one of the affections in which the influence of heredity is most obviously seen, particularly in families in which other evidences of "scrofula" or of the "strumous" diathesis exist. Various exanthems, for instance, measles, scarlet fever, and diphtheria, strongly dispose to it, whilst in many cases it results from repeated attacks of tonsillitis. The enlargement may date from infancy or occur at puberty. Large hypertrophy is rare after thirty-five; and the tonsils, if enlarged in childhood, tend to atrophy at puberty: although it is important to know that this rule is by no means without exception. In many cases there is no obvious cause for the condition; but in most there is a combination of several of the above contributory factors. Very frequently it is found associated with hypertrophy of the pharyngeal tonsil (adenoid vegetations), and, not quite so often, with enlargement of the lingual tonsil and of the cervical lymphatic glands.

Pathology.—The substance of the healthy tonsil is composed of a number of small nodules of lymphatic tissue arranged around a group of seven to twelve crypts, and of connective tissue, blood-vessels, and a few nerve fibres; the tonsils are covered by ordinary mucous membrane which dips down into the crypts. There are no secretory ducts, nor does the mucous membrane even in the crypts present any appearance of muciparous glands; but leucocytes pass out through minute spaces between the epithelial cells, and the mucous membrane is capable of secreting small quantities of mucus. In the large soft chronic hypertrophy of the tonsils, such as is generally seen in young patients, the lymphatic nodules are increased in size and number, and the gaping crypts contain a variable amount of mucus and of altered epithelium undergoing fatty degeneration. In other cases, chiefly in adults, the hypertrophy is mainly due to an excessive growth of the connective-tissue elements, which, by compression, cause more or less atrophy of the lymphatic nodules and blood-vessels, and obliteration of the crypts; changes which result in a hard, smooth, non-vascular tonsil.

We distinguish three clinical varieties :—(i.) *Chronic lacunar tonsillitis* with accumulation of caseous matter in the crypts, which gape when the yellow evil-smelling masses are extruded. These masses are sometimes very consistent, and may be confused with keratosis, but examination of the very adherent, clear, milky-white, opaque, soft, projecting pointed masses of the latter will reveal the characteristic threads of the cryptogam. (ii.) *Chronic parenchymatous hyperplasia.* The tonsils are soft and friable from the overgrowth of lymphoid tissue. (iii.) *Chronic fibroid degeneration.* This form is almost confined to adults, it represents the advanced stage of the hyperplastic form, it is often the remnant of former frequently occurring attacks of acute tonsillitis, and it is especially associated with the rheumatic or gouty habit.

Sometimes we meet with a smooth, pale yellowish swelling due to occlusion of the mouth of a crypt with retention of the cheesy exudation —a form of chronic tonsillar abscess.

The enlargement of the tonsils is sometimes very great, occasionally enormous, projecting far beyond the palatine arches, and meeting in the middle line behind the uvula. Not infrequently the tonsil becomes adherent to the anterior pillar of the fauces, which may then extend over the whole anterior surface, completely concealing the tonsil itself.

Symptoms.—Owing to the enlarged tonsils encroaching on the oropharyngeal space, and interfering with the movements of the soft palate, the voice is throaty and thick, with a nasal twang. Pain is generally absent except in subacute attacks of tonsillitis, which generally occur at frequent intervals. In children especially, in whom most of the cases are met with, post-nasal adenoids are generally present also, and many of the symptoms attributed to enlarged tonsils—such as anaemia, buccal respiration, pigeon-breast, and infra-mammary depression of the ribs, small ill-developed lungs, snoring, suffocative symptoms, and night-terrors during sleep, difficulty in deglutition, and particularly Eustachian deafness—are in the main due to the concomitant adenoids; though most of these symptoms may be due to the tonsillar disease alone, without adenoids. The mouth is often kept open, the under lip protruding; and thickening behind the angle of the jaw and enlargement of the cervical lymphatic glands are frequently present. Dry reflex cough is a very common symptom, and various reflex neuroses, such as darting pains in the ears, vomiting, and gastric pains, have been attributed to enlarged tonsils. The constantly recurring attacks of tonsillitis, in addition to the suffering they entail, are attended by high fever and followed by great prostration; thus they greatly interfere with occupation, development, and general health.

Chronic enlargement of the tonsils may then act injuriously in three different ways—namely, (*a*) by mechanically obstructing the food and air passages; (*b*) by maintaining a liability to frequent, often very painful attacks of inflammation within the glands themselves or in their immediate neighbourhood, and by the chronic toxaemia from the contamination of the blood circulating through the tonsil, causing the so-called "poor circulation" or lack of normally active tissue-metabolism so often seen in patients with enlarged tonsils; (*c*) by forming a perpetual source of danger from infection by various micro-organisms, such as those of diphtheria or tuberculosis.

The prognosis, as regards the life of the patient, is invariably good; the ultimate effect on the health of the patient will depend to a certain extent on his age, on the relative degree of hypertrophy of the tonsils as compared with the size of the fauces, and on the presence or absence of concomitant adenoid vegetations in the rhino-pharynx.

In children under the age of ten marked hypertrophy greatly interferes with growth and healthy development; and the coexistence of adenoids adds to the pernicious effects that will almost certainly ensue to his permanent disadvantage. A tonsil not excessively hypertrophied may undergo the physiological retrogressive changes soon after the age of puberty; but in most cases we shall await such a happy consummation in vain, and meanwhile the child is exposed to the numerous risks already described.

From a therapeutic standpoint the prognosis is excellent provided no irremediable consequences have ensued; thus, the prospect of perfect recovery depends on the absence of marked deformity of the chest walls and other secondary changes.

Treatment.—The only satisfactory method of dealing with enlarged tonsils which require treatment is to remove them; and at the outset we would emphasise the uselessness of the so-called milder measures, particularly the ridiculous painting with iodine solutions, tannic acid, and the like; these prescriptions are so much waste of time, and generally succeed only in causing considerable annoyance to the patient.

The tonsils should be reduced in size, (*a*) if they interfere with respiration, either during waking or sleep, and lead to deficient aeration of the blood ; (*b*) if they lead to changes in the character of the voice and to defective articulation ; (*c*) if they lead to defective development of the face and chest ; (*d*) if the chronic enlargement, though not very considerable, be attended with frequent attacks of inflammation of the tonsils themselves, by tumefactions of the cervical glands, or by catarrhal conditions of the neighbouring mucous membrane, especially of the Eustachian tubes : even in the absence of symptoms, decided chronic hypertrophy, especially in association with the "strumous diathesis," renders an operation advisable in patients under fourteen, so that a very active source of danger from infection may be removed.

Removal of the tonsils may be accomplished by various methods : by cutting with the bistoury or tonsillotome, by enucleation, by the galvano-caustic point or snare, or by punching out. Our own practice is confined to two methods, namely, tonsillotomy and the galvano-caustic point. When it has been decided to remove the tonsils we have to consider which is the best method to choose. (α) If the patient be under twenty, and the enlargement be mainly transverse, so that the tonsil or tonsils project a good deal beyond the arch of the palate, the cutting operation should be preferred. (β) Local conditions being the same, but the patient over twenty years of age, and in all cases in which the tonsils are entirely concealed behind the palatine arches, or only project a little beyond them, let broad applications of the galvano-cautery be made by means of a large flat burner of platinum or porcelain. The tonsils are reduced in size by the cautery quite as rapidly and effectually by this method as by galvano-puncture ; and it has the advantage of greatly reducing the risk of sharp haemorrhage, a risk which cannot altogether be disregarded. The cutting operation in patients over twenty is more liable to be attended with serious and uncontrollable haemorrhage than is the case in younger patients.

The object of the operation should be to reduce the tonsil to the normal size ; and therefore in using the tonsillotome it is well at the moment of performing the operation to push the tonsil a little inwards by firm pressure from without just underneath the angle of the jaw, so that the portion of the tonsil lying between the palatine arches is removed without either injuring the anterior arch of the palate or running a risk of injury to the large vessels in close relation with the base of the tonsil. Sometimes the palatinal arches when adherent to the tonsils may have to be freed from them by means of a blunt hook introduced between the arches and the anterior surfaces of the tonsils previous to the removal of the latter. It is useless to remove a superficial slice in the hope that the remainder will atrophy.

When the galvano-cautery is employed, the reduction of the tonsils will require six, eight, or ten sittings, according to the degree of enlargement, at intervals of three days to a week. The amount of reduction will have to be determined on the merits of each case.

Removal is most readily accomplished by the tonsillotome. One of us (F. S.) employs Mackenzie's instrument; by the other (W. W.) Matthieu's tonsil guillotine is always preferred. In young and nervous children the operation may be done under ethyl chloride, ether, or chloroform administered as in operating on post-nasal adenoids. Again we wish to emphasise the rule that in all cases of enlargement of the tonsils the existence of adenoids should be investigated; and, if present, they should be removed first. In adults and in older children, when the tonsils only require removal, nitrous oxide gas anaesthesia suffices, or a general anaesthetic may be dispensed with, and a strong solution of cocaine, alypin, or eucaine used instead to produce local anaesthesia. After operation the patient should be directed to keep quiet for a few days, and only bland, cold, and soft food should be taken.

Haemorrhage is always pretty free after tonsillotomy, but usually ceases spontaneously in a few minutes. Dangerous haemorrhage occurs in a very small percentage of cases, however skilfully the operation is performed; yet so rarely in proportion to the number of operations, that it can never be urged as a general objection to the practice. In children it is extremely rare; and it is in the older patients whose enlarged tonsils have undergone cicatricial degeneration that haemorrhage is to be feared, and this more especially after cutting operations.

The causes of haemorrhage may be stated briefly as (*a*) abnormality in the distribution of the blood-vessels; (*b*) fibroid degeneration of the tissues and of the walls of the vessels, which gape when they are divided; (*c*) haemophilia; (*d*) eating solid food; and (*e*) over-use of the voice too soon after the operation.

If the haemorrhage does not soon cease spontaneously, or if secondary haemorrhage occur, the patient should be kept quiet and have small pieces of ice to suck, and a mixture of tannic and gallic acids dissolved in water to sip; or the solution may be applied directly to the bleeding tonsil. Perchloride of iron, which is sometimes recommended, should never be applied, as it produces clotting without arresting the haemorrhage, conceals the bleeding spot, and often enough makes matters worse by inducing retching from the mechanical irritation of the fauces produced by the blood-clots. Application of peroxide of hydrogen, on the other hand, may prove useful. If ordinary styptic measures fail, we must seek for the bleeding point, and, if possible, the vessel from which the haemorrhage flows should be seized and twisted with torsion forceps; or the sources of haemorrhage may be touched with the galvano-cautery. Direct compression may be made by means of a tonsil forceps or clamp, a simple and easily applied form of which is illustrated (Fig. 26). The wide distal ends are firmly wrapped in cotton wool and one end covered with tannic and gallic acid paste is placed over the bleeding tonsil, while the other blade is outside; any necessary degree of pressure is obtained on closing the blades, which are retained in position by a ratchet. It may be possible with forceps or tenaculum to draw the remains of the tonsil inwards beyond the faucial pillars and to pass a ligature round the bleeding point, or a ligature may be passed through the upper part of the stump of the tonsil from before backwards and brought back through the lower border and then tied so as to compress any remaining portion of the tonsil. But if the tonsil has been so completely removed that there is no stump, and other means have failed, the method of suturing advocated by Douglass may be resorted to. A specially devised instrument, such as Douglass's tonsil needle or Horsford's epiglottis needle, may be used, or in an emergency a Hagedorn needle firmly fixed in a needle-holder will serve the purpose. Threaded with heavy silk, the needle should be passed from behind forwards through the posterior faucial pillar, then through the upper part of the base of the tonsil, and then through the anterior faucial pillar. This needle will transfix the posterior pillar, the body of the tonsil, and the anterior pillar, and should emerge from the anterior pillar a quarter of an inch from its edge. It is then drawn through, bringing with it the silk, the two ends of which are secured and held by an assistant. Similar sutures are then made in the middle and the lower part of the tonsil. When two or three sutures have thus been introduced they may be tied firmly so as to fold half of the cut surface of the tonsil upon the other half. The sutures may be removed about the third day. Direct persistent digital compression has sometimes to be resorted to, and if even this fail, ligation of the carotid arteries, especially of the external carotid, remains as our last resource.

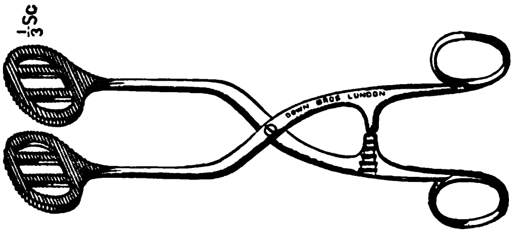

FIG. 26.—Watson Williams's tonsil compressor.

There still exists in the minds of the public and even of many practitioners a prejudice against operations on the tonsils; it is necessary, therefore, to refer briefly to some of the objections raised. First, it is urged in the case of children that they will "grow out of it," and that if matters are left to nature the tonsils will spontaneously atrophy at puberty or soon after that time. It is true that in a certain proportion, about two-thirds of the cases, the tonsils, by the age of twenty, either atrophy or cease to be inconvenient enough to urge the patient to seek advice; but in the remaining one-third of the cases this spontaneous disappearance does not occur, and therefore, although we may certainly tell the parents of a child suffering from enlarged tonsils that there is a chance of their atrophy after puberty, we must warn them that this event is by no means certain. But suppose our best hopes fulfilled, we have still to consider the great risks of serious and lasting consequences of great hypertrophy of the tonsils during the earlier years of life. If a child has not begun to suffer from the consequences of obstruction to the respiratory, alimentary, and auditory passages till the age of ten (and nearly half the total number of cases display marked symptoms before that age), and if we admit that his tonsils may atrophy by the age of eighteen, can it be fairly urged that eight years of constant interference with some of the most important functions of life, and that during the most important period of development, will not leave behind them lasting injury? The number of adult patients suffering since childhood from "throat deafness," and gradually getting worse, the configuration of countless faces seen in the streets, the defective articulation and intonation so often met with in people in society—all these defects tell their own tale and give the best reply to the question. And even if such sequels do not follow, the patient is liable to frequently recurring acute attacks of throat disorder.

Further, it is sometimes stated that the tonsils, even when hypertrophied, are protective against infectious disease; now it has been conclusively proved that tonsillar hypertrophy adds very greatly to the danger of infection, a point which we have fully emphasised in our opening remarks on diseases of the tonsils.

Removal of the tonsils never impairs the voice; on the contrary, in cases in which the masses of hypertrophied tissue are large enough to interfere with the normal vibrations of the column of air, and to divert it into an anomalous direction, and at the same time to interfere with the movements of the palatine arches and soft palate, and perhaps to maintain a chronic catarrhal pharyngitis, the voice will certainly be greatly improved in strength, quality, and timbre; although the removal of the tonsils will not of itself increase the range of the voice.

That removal of the tonsils has any tendency to result in sterility is a superstition so absurd that it is only worth mentioning to shew that no belief is too foolish and groundless to be advanced against tonsillotomy.

In cases which urgently call for operative interference, not only are all the risks of local complications due to the enlarged tonsils removed, but there is almost invariably a rapid and marked alteration for the better in general health and development where these have been impaired. The appetite and digestion are improved, there is better aeration of the lungs, the child becomes fat, rosy-faced, bright and cheerful, and is a marked contrast indeed to the half-nourished, listless, anaemic, more or less deaf creature with open mouth and noisy respiration. The operation should not be postponed on account of the weakly condition of the patient; for though it seems reasonable to suggest that it would be well to wait until a course of careful dieting and general treatment have made the child stronger and better able to undergo operative treatment, we should remember that the local conditions are in themselves chiefly responsible for the adverse state of health, and that until the tonsils are removed but little amelioration can be anticipated; whereas the tonic treatment which has usually been tried before and failed will be attended with very much happier result after the operation, or, indeed, is usually rendered unnecessary thereby. We have never in the whole of our experience seen any benefit derived from a postponement of the operation in the class of cases now under discussion.

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Tonsilloliths.—Tonsillar Calculi, Calcareous Concretions.

Etiology and Pathology.—Tonsilloliths usually vary in size from quite small concretions, in which case they are usually multiple, to the size of a filbert, though they sometimes attain much greater dimensions, the larger calculi being generally single. Dr. Aitchison Robertson records and figures one that weighed nearly an ounce; it measured $1\frac{3}{4}$ by $1\frac{1}{2}$ inch.

In the great majority of cases calcareous concretions in the tonsils are originated by the *Leptothrix buccalis* or other organisms in the tonsillar crypts, in much the same way as broncholiths are formed or as tartar becomes deposited on neglected teeth. Around this nucleus altered mucus, pus, and epithelial cells collect and become calcareous. In this manner several accumulations of calcareous matter may come to occupy the crypts; or one or more large calculi, varying in size up to more than an inch in diameter, may be formed. It is in the upper part of the tonsil or in its deeper aspects that the calculus is generally formed, and it seems probable that it is due to the accumulations in a partly closed supratonsillar fossa, or in the deeper ramifications of the fossa in the manner suggested by Dr. Paterson. Dr. Robertson suggests that the larger calculi probably result from the retention of pus in the interior of the tonsil, the residual matter undergoing caseation and subsequent calcification. Dr. H. Walsham considers that at least some tonsil calculi are originated by those epithelial pearls which frequently occur in sections of the tonsil and which are retention products.

The crypt affected is gradually distended, and may then occupy a considerable area within the tonsil. It has been affirmed that they are of gouty origin, and very rarely "chalk-stones" of urate of sodium have been observed in the tonsil. But what are usually known as tonsilloliths consist chiefly of phosphate and carbonate of lime. Dr. Robertson gives the subjoined analysis of tonsillar calculi:—Organic matter, 18·40 per cent; inorganic, 81·60 per cent; phosphoric anhydride, 50 per cent; calcium and magnesium oxides, 21·20 per cent.

Symptoms.—The symptoms are often very slight, and are simply those common to enlarged tonsils, whilst occasionally persistent pain is complained of: sometimes a calculus maintains a certain degree of chronic inflammation. If the tonsillolith sets up suppurative inflammation, it may be extruded with the rupture of the abscess, and the symptoms subside. But when this occurs with a large calculus there is a danger of its suddenly lodging in the larynx, as happened in Dr. Robertson's case, with nearly fatal asphyxia.

The *diagnosis* may be made by means of a probe, or, in the case of larger deposits, by palpation.

The calculus should be removed, and if the tonsil be hypertrophied, or multiple small concretions be present, it is better to remove the gland altogether.

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Diseases of the Lingual Tonsil.—The fourth tonsil situated at the base of the dorsum of the tongue is liable to the same diseases as the palatine tonsils. Thus, it may be the seat of acute lacunar or parenchymatous inflammation which may go on to suppuration, or of tuberculosis, syphilis, and benign or malignant growths. Occasionally aberrant thyroid gland tissue forms distinct tumours in the region of the lingual tonsil (*vide* Vol. III. p. 325).

Chronic hypertrophy is frequently found in a mild degree in chronic pharyngitis, and more marked—often without adequate explanation—in otherwise healthy persons, particularly in women. In the last-named class of cases, by direct contact of the hypertrophic glandular tissue with the dorsum of the epiglottis, it often gives rise to a constant irritating cough, sensations of fulness, choking, "lump in the throat," and so forth. Many cases of so-called globus are of this kind. The hypertrophied tissue is sometimes seen quite to overlap the epiglottis, often, indeed, almost to conceal it

The hypertrophy should be reduced by applications of Lugol's solution (iodine grs. xx, iodide of potassium grs. xxx, to an ounce of water); or by the lingual tonsillotome, curette, or snare according to the shape and size of the mass. The employment of the galvano-cautery, which is often recommended, is not free from the risk of causing violent parotitis. An aberrant thyroid gland occupying the position of the lingual tonsil should not be removed, even if a source of inconvenience, unless it has been ascertained that the thyroid gland is not absent in the neck, otherwise myxoedema may supervene. This occurred in one case (*vide* also Vol. III. p. 325).

FELIX SEMON.
PATRICK WATSON WILLIAMS.

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F. S.
P. W. W.

Foreign Bodies in the Air- and Upper Food-Passages.—This subject is discussed in connexion with the articles on "Oesophagoscopy," "Tracheoscopy," and "Bronchoscopy" (p. 299), and on "Diseases of the Trachea" (p. 328).

III.—DISEASES OF THE LARYNX

- Laryngoscopy
- Special Methods of Laryngoscopy.
- Anaemia and Hyperaemia.
- Laryngitis—
  - Acute.
  - Chronic.
- Oedema.
- Haemorrhage.
- Tuberculosis.
- Lupus of the Pharynx and Larynx.
- Leprosy of the Pharynx and Larynx.
- Myxoedema.
- Acromegaly.
- Syphilis.
- Perichondritis.
- Diseases of the Crico-Arytaenoid Joint.
- Stenosis.
- Benign Growths (including Pachydermia).
- Malignant Growths.
- Neuroses.
- Paralysis.

PLATE VI.

THE NORMAL LARYNX.

Fig. 1. The laryngoscopic image, shewing the position of the vocal cords during quiet respiration.

,, 2. The same on deep inspiration, with the vocal cords fully abducted.

,, 3. The same on phonation, the vocal cords in the position of adduction.

Figs. 4 and 5. The same, shewing the vocal cords in the cadaveric position—Fig. 4 the minimum, and Fig. 5 the maximum separation of the vocal cords.

Fig. 6. A normal female larynx, the vocal cords adducted in phonation.

,, 7. A larynx during phonation. In this case the cartilages of Santorini and the arytaenoids of each side overlap. (From a drawing by the late Sir Morell Mackenzie.)

III.—DISEASES OF THE LARYNX

By Sir Felix Semon, K.C.V.O., M.D., F.R.C.P., and P. Watson Williams, M.D.

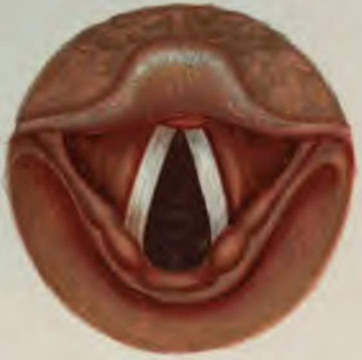
Laryngoscopy

Inspection of the Larynx.—For this purpose a small mirror attached to a handle must be introduced into the back of the mouth, and a strong light thrown on the reflecting surface, which is directed obliquely downwards so as to reflect the image of the larynx. The small laryngoscopic mirror should be attached to the handle at an angle of about 120°. At least three sizes of these flat circular mirrors are desirable, of diameters of half an inch, one inch, and 1½ inch respectively, adapted, that is, to the size of the fauces at different ages.

The forehead reflecting mirror is concave and of about fourteen-inch focus. It should be adjusted, by a freely adjustable ball and socket joint, to a forehead band or spectacle-frame carrier; the latter has the advantage of being more readily put on and off, and for hypermetropic or myopic observers spectacle glasses can be attached to the frame. In the centre it should have an oval opening, the long axis of which corresponds with the long axis of the observer's eye. It is essential that the central opening should come immediately in front of the pupil of the examiner's eye, and that the mirror be freely adjustable.

For a satisfactory examination a good light is of the utmost importance. Bright sunlight answers admirably when it is available; but it is usually more convenient to employ some form of artificial light which is wholly under control. In a darkened room a candle, an Argand burner, or oil lamp may suffice in some cases for diagnostic purposes; but a high-candle-power incandescent burner, or an electric lamp, for which the Nernst light is best, or the oxyhydrogen lime-light is desirable for finer operations and for higher degrees of accuracy of diagnosis in many of the less gross or more obscure laryngeal affections, and for posterior rhinoscopy. The artificial light should be freely movable in every direction, so as to allow of ready adjustment and focussing of the light on the part to be examined. The examiner should also accustom himself to the use of ordinary bright daylight concentrated by the forehead mirror upon the patient, whose back is to a window, as this may give a better

PLATE VI.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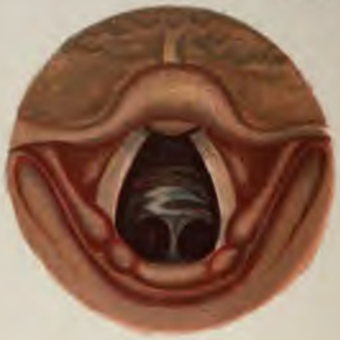
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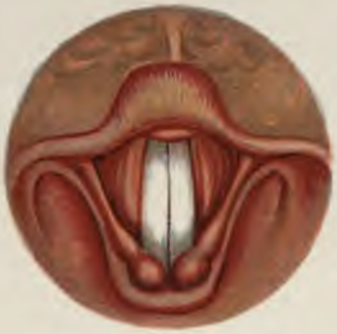
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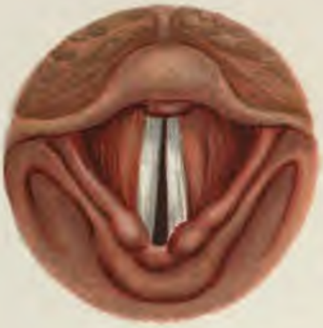
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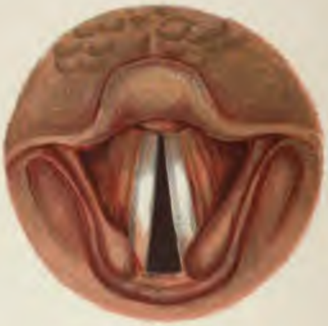
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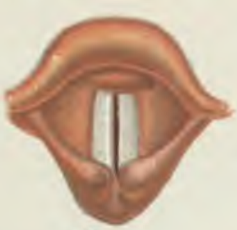
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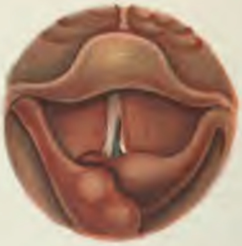
Fig. 7.

arytaenoid space or fold forming the posterior wall of the larynx. The folds of mucous membrane stretching on each side between the epiglottis and the arytaenoid cartilages are the arytaeno-epiglottidean folds; and posteriorly, just in front of the arytaenoid cartilages, the cartilages of Wrisberg and Santorini can often be recognised in the outline of these folds. Between the arytaeno-epiglottidean folds and the prominence of the great cornu of the hyoid bone are the pyriform sinuses or hyoid fossae.

In making a laryngoscopic examination we first observe (*a*) the colour of the various parts; secondly (*b*), the form and contour; and lastly (*c*), the functional activity of the vocal cords during phonation and respiration. As regards the colour, the epiglottis should be slightly yellowish and the rest of the laryngeal mucous membrane pale pink or red, whilst the vocal cords are, normally, pearly white or very slightly pink, though they are often of a more pronounced reddish colour, particularly in male professional vocalists. The vasomotor changes in the larynx are very rapid: on first introducing the mirror, anaemia may be present; this on a second inspection may have given place to the normal tint, and on the third to hyperaemia. As isolated anaemia of the larynx is a valuable diagnostic sign in tuberculosis, this inconstant condition should be carefully noted on the first inspection, whilst the structural alterations and the movements of the vocal cords may be left to a later observation. The structural alterations to be noted are tumefaction, ulceration, abscess, oedema, new growths, foreign bodies, malformations, and dislocations of the arytaenoid cartilages. Any uneven margin or surface of the vocal cords should be particularly noted. Finally, the position and motility of the cords will engage attention. No definite conclusion concerning the motility of the vocal cords can be gained, unless the larynx be examined both during phonation and deep inspiration. The neglect of this fundamental rule often results in overlooking laryngeal paralysis. During quiet respiration the vocal cords should lie midway between adduction and abduction, "the position of rest or quiet respiration"; this is not the same as the "cadaveric" position in which the glottic chink is narrowed, for the wider aperture of rest, as has been shewn by one of us (F. S.), is maintained by a persistent reflex tonus of the abductors. On phonating "eh! eh!" the vocal cords should come into symmetrical apposition in the middle line; the arytaenoid cartilages at the same time being approximated by the arytaenoideus muscle so as to obliterate the interarytaenoid space. During deep inspiration the cords are widely abducted, so that the glottic opening and the interarytaenoid space are considerably wider than during quiet respiration. It is not enough simply to observe that the vocal cord moves out on taking a breath; it is important to note also whether the degree of abduction on deep inspiration amounts to the normal.

There are then four defined positions of the vocal cords, namely, those of (α) quiet respiration, (β) deep inspiration, (γ) phonation, and (δ) the cadaveric position of death or complete paralysis. Examples of these various positions are illustrated on Plate VI.

Finally, it may be necessary to test the tactile sensibility of the larynx by means of a long curved laryngeal probe. The normal larynx is very sensitive, and on contact violent cough is immediately set up, particularly when the inter-arytaenoid fold is touched. In anaesthesia this sign is absent.

Difficulties in laryngoscopy may be encountered; sometimes these are due to the faulty method of the examiner, sometimes to structural peculiarities in the fauces or larynx of the patient. The following faults should be avoided: undue haste, flurrying the patient and rendering him nervous, attempts at examination without having the light properly concentrated; clumsy introduction of the mirror, or introduction of a mirror either not properly warmed or made too hot; dragging on the tongue or pressing it against the lower incisors; omission to tell the patient to breathe quietly and naturally; holding the mirror too long in the mouth, and neglect of the various little manoeuvres for bringing the larynx into view by getting the patient to tilt his head backwards or forwards as may be required.

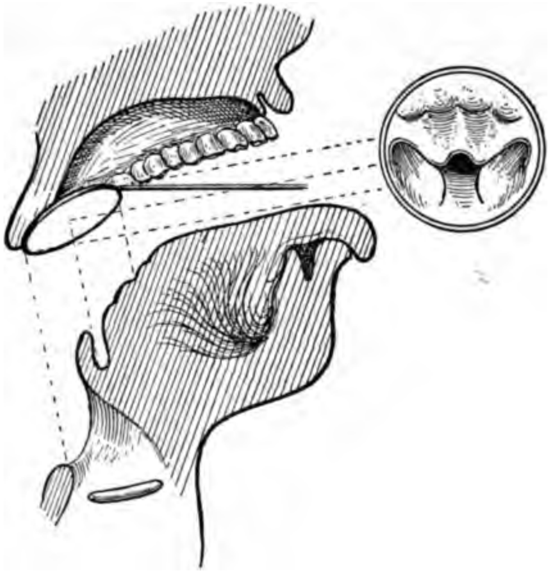

FIG. 28.—Diagram shewing the mirror held too far forward and in too horizontal a position to gain a view of the vocal cords; the upper surface of the epiglottis and the base of the tongue coming into view in the laryngeal mirror.

A common fault is to hold the laryngoscopic mirror at the wrong angle, or too far forward, so that only the dorsum of the tongue and the anterior surface of the epiglottis are reflected in it (Fig. 28). By placing the mirror somewhat farther back and less horizontally, a complete image will probably be obtained.

Difficulties may arise on the side of the patient. Of these the most common are: (α) Excessive irritability of the fauces, leading to gagging and retching on the introduction of the mirror. To overcome this the patient may suck ice for fifteen or twenty minutes before the examination, or a 10 per cent solution of cocaine, novocaine, alypin, or stovaine may be applied to the fauces. In very irritable subjects it may be necessary to apply the solution, not only to the anterior surface of the soft palate, but also to the posterior wall of the soft palate and corresponding portion of the posterior pharyngeal wall, otherwise the introduction of the mirror merely presses the soft palate back against the irritable pharyngeal mucosa. (β) The dorsum of the tongue may rise so much that either the mirror cannot be introduced, or its reflecting surface is out of view. If forcible protrusion of the tongue by the patient or taking a deep breath does not overcome this difficulty, the patient should be asked to hold his own tongue, while the examiner depresses it with a tongue spatula held in the left hand. Sometimes the best view is to be had by simply depressing the tongue without protrusion; and if the patient be tongue-tied or protrusion impossible, this procedure should be adopted in the first instance. (γ) The tonsils may be so enlarged that the usual mirror cannot be used; in these cases it may be possible to introduce a smaller one. If the uvula be excessively long, it may get in the way; this obstacle will be overcome by using a large mirror. (δ) The most serious difficulty is a pendulous epiglottis so overhanging the larynx that the anterior portion of the larynx is concealed from view, and perhaps nothing but the posterior border is reflected. There are several ways of overcoming this difficulty. In slighter cases the act of phonating "ēē! ēē!" or coughing with the mirror in place, may suffice to raise the epiglottis; then the vocal cords may come into view. If this manoeuvre fail, direct the patient to throw his head well back, and place the mirror nearer the posterior wall of the pharynx, and somewhat more vertically than usual, the observer's eye being well above the level of the patient's mouth. In a few very exceptional cases, however, it is only possible to see the vocal cords by raising the epiglottis with a retractor or by passing a thread through it by means of a specially constructed instrument. A very ingenious instrument for this purpose has been recently constructed by Dr. Cyril Horsford. (ε) The patient may hold his breath from nervousness; but a little patience will soon overcome this difficulty. When nervous patients close their eyes, not only do they become more nervous, but they tend also to hold the breath, for closing the eyes usually goes hand in hand with holding the breath, hence they should be told to keep their eyes open. It is important to remember that in nervous patients the vocal cords, instead of being widely abducted on deep inspiration, may be partially adducted, so that to the careless or inexperienced observer they may appear to be affected with paresis of the abductors.

The chief *congenital defects* that are met with are a deep central notch in the free border of the epiglottis, which may extend so far downwards as to produce a bifid or double epiglottis; and a membranous web between the vocal cords, which in some cases extends backwards as far as the vocal processes (Fig. 1, Plate VII.). In a case observed by one of us (F. S.) the web was associated with coloboma iridis; the web being so thick that it proved impossible to divide it with a Mackenzie's laryngeal knife, and necessitated repeated puncture with a galvano-cautery point for its successful removal. These webs probably arise from the incomplete absorption of the tissues between the epiblastic cul-de-sac and that of the hypoblastic air-tube in the region of the larynx. Bruns cites observations on the embryonic development of the larynx in support of this opinion. That the tendency to the formation of these webs may be hereditary is shewn by a series of cases in a family recorded by Seifert. Another congenital defect is the incomplete union of the alae of the thyroid cartilage in front; an instance of this was demonstrated many years ago by one of us (F. S.), the gap being filled in by a fibrous web.

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Special methods of laryngoscopy are at times called for and require a short description.

(*a*) *For inspection of the anterior surface of the inter-arytaenoid fold of either arytaenoid eminence* the Killian position is sometimes invaluable. The patient stands and looks down towards the examiner, whose head is on a level with the patient's epigastrium and looking up. The mirror is then introduced as in ordinary laryngoscopy, the source of light being arranged accordingly. The anterior half of the larynx is then generally concealed by the epiglottis, but the much more direct view obtained of the parts around the posterior half of the glottic aperture may reveal an ulcer or tumefaction which was quite out of view with ordinary laryngoscopy.

(*b*) *For inspecting the posterior external surface of the larynx*, especially that portion lying in contact with the laryngo-pharyngeal wall, and particularly valuable for inspection of the laryngo-pharynx generally, resort may be had to *hypopharyngoscopy*, a term introduced by von Eicken (*vide* p. 317). Following him Gerber has devised an angular spatula for pharyngo-laryngoscopy, the distal end of which has a downward direction which is introduced behind the larynx and then drawn forwards, somewhat after the method of Blumenfeld. Von Eicken's method is probably more efficient. The fauces and larynx having been carefully rendered insensitive with cocaine or one of its substitutes, the patient is seated in a high chair, and the examiner kneels before him, so that the patient during examination inclines his head forwards and downwards. This not only enables the examiner to get a good view of the posterior surface of the larynx, but ensures also the relaxation of the extrinsic muscles which interfere with the due retraction of the larynx. A specially stout laryngeal probe is then introduced through the glottis into the subglottic space, guided by the laryngoscopic mirror in the usual way. The larynx is then firmly pulled forwards and upwards away from the posterior surface of the pharynx, from which it may thus be separated from one and a half to two centimetres, allowing the inspection of the parts thus exposed. Hald of Copenhagen finds it useful to draw a thin indiarubber tube over the distal part of the laryngeal tractor to protect the subglottic mucosa. This observer states that he has not rarely seen the gullet open up during vocalisation, giving an opportunity of inspecting the upper part of that canal.

Concerning the more recently introduced methods of direct inspection of the larynx, such as Kirstein's "autoscopy" and Killian's or Brüning's tubes for examination, the reader is referred to the article on "Bronchoscopy" (see p. 299).

Skiagraphy of the Larynx.—Röntgen's *x*-rays are very useful for the discovery of those foreign bodies impacted in the larynx which are impenetrable to these rays, as stated in the section on foreign bodies in the upper air- and food-passages (*vide* p. 328).

Finally, we would emphasise the great importance of bearing in mind that in laryngeal affections, as in all local maladies, due regard must be paid to the general condition of the patient, to his facial aspect, his gait, and the state of his pulse, heart, lungs, and so forth. Neglect of this fundamental rule may lead to the gravest errors in diagnosis; thus, acute laryngitis may be due to gout, or recurrent attacks of laryngitis to early pulmonary tuberculosis; whilst a persistent and troublesome cough may be the earliest manifestation of tabes dorsalis; not to mention the grosser laryngeal lesions that may baffle diagnosis unless the facts of the previous history and of a general examination of other regions are taken into consideration, for example, in syphilitic disease.

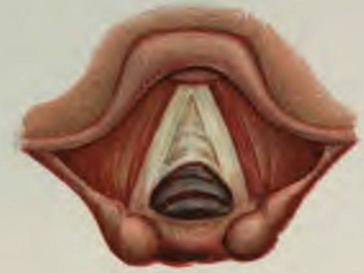
Anaemia.—The larynx shares in the general pallor of the mucous membranes which is seen in anaemia. Isolated anaemia of the larynx is not infrequently the precursor of laryngeal tuberculosis; it is therefore an indication which should be carefully watched.

Hyperaemia.—All inflammatory states of the larynx are preceded by hyperaemia; hence hyperaemia of the larynx is in general an indication of the catarrhal process. One exception, however, deserves attention, namely, that in a certain number of men who constantly use the voice, as in singing, the vocal cords become slightly hyperaemic without in any way affecting the purity of the voice.

ACUTE AND CHRONIC INFLAMMATORY DISEASES OF THE LARYNX

Acute Laryngitis.—Acute catarrhal laryngitis in its milder forms, being one of the commoner minor ailments that are frequently associated with

PLATE VII



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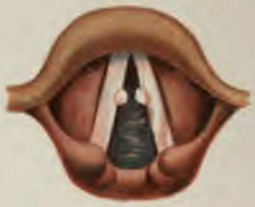

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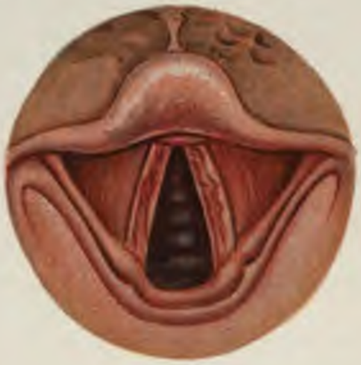

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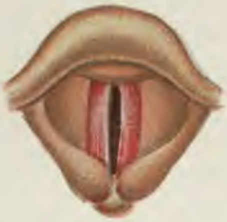

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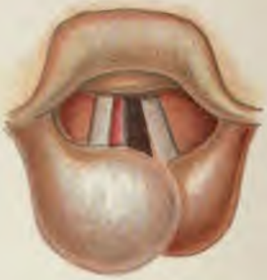

Fig. 5.

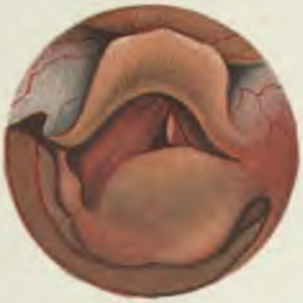

Fig. 6.

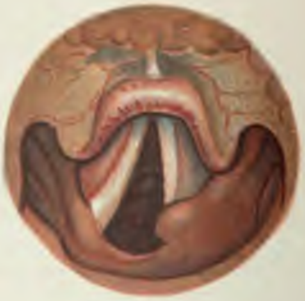

Fig. 7.

PLATE VII.

INFLAMMATORY DISEASES OF THE LARYNX.

Fig. 1. Congenital web between the anterior two-thirds of the vocal cords. A somewhat similar web may arise in after life as the result of syphilitic or other disease, traces of former disease in other parts of the larynx being then present in the form of cicatrices, etc. (P. W. W.)

„ 2. Singer's nodes (chorditis tuberosa). (P. W. W.)

„ 3. Haemorrhagic laryngitis. (P. W. W.)

„ 4. Laryngitis. The vocal cords are red, and do not come into apposition on phonation owing to myopathic paresis of the internal thyro-arytaenoid muscles. (P. W. W.)

„ 5. Inflammatory oedema of the right arytaenoid mucosa. Infraglottic oedematous infiltration is also seen beneath the right vocal cord. (P. W. W.)

„ 6. Perichondritis and oedema of the left arytaenoid. (Krieg.)

„ 7. Ankylosis of the left arytaenoid cartilage from inflammatory thickening in and around the crico-arytaenoid joint. (Krieg.)

an ordinary cold in the head, is too familiar to need lengthy description, but it is a serious consideration to professional voice-users, and its bearing on persistent impairment of the voice must be borne in mind. In children, too, with a normally small glottic aperture, the more severe attacks of catarrhal laryngitis may cause severe distress and even fatal dyspnoea.

Etiology and Pathology.—The usual exciting causes are (*a*) exposure to sudden changes of temperature, especially in association with damp climatic conditions; (*b*) over-use or straining of the voice, as in loud shouting, or prolonged vocal efforts with faulty production; (*c*) inhalation of irritating vapours, very hot steam, irritating particles of dust; (*d*) gout or rheumatism. Acute laryngitis, indistinguishable from simple catarrhal laryngitis, is one of the commoner symptoms of influenza, measles, German measles, scarlet fever, small-pox, and more rarely occurs in syphilis, tuberculosis, enteric and typhus fevers, and many other general diseases (*vide* p. 286 *et seq.*).

As disposing causes we may mention sedentary habits, alcoholic intemperance, excessive smoking, living in over-heated rooms, and any conditions which render patients susceptible to changes of temperature, whilst marked nasal obstruction, with resulting buccal respiration, undoubtedly renders the individual prone to attacks.

The morbid appearances differ but little from those seen in any acute catarrhal inflammation of the mucous membranes generally, but is most striking in the heightened colour of the epiglottis and redness of the normally white vocal cords. Their superficial layer of epithelium is largely shed, and the submucosa is slightly infiltrated and swollen. But the inflammation extending to the superficial muscles impairs their movements and renders them readily liable to overstrain; such myositis is perhaps the chief cause for a lasting impairment of voice from its over-use during even a slight laryngitis. It may be sufficiently marked to result in paresis of the thyro-arytaenoidei interni (Fig. 4, Plate VII.).

The *symptoms* vary, inasmuch as they depend largely both on the acuteness and severity of the attack, and the age and sex of the patient. In adults the symptoms are either purely local or at most associated with slight malaise and a very moderate rise of temperature; in other words, with the symptoms of a severe cold. Soreness is usual in the laryngotracheal region, especially on attempting to use the voice, and the voice is hoarse or more or less completely aphonic. These symptoms are accompanied by a dry tickling cough, with little or no expectoration, unless the inflammation extends to the trachea and bronchi. Sometimes after severe coughing a small pellet of greyish mucus is expectorated, and at times this may be streaked with blood. Considerable pain may be experienced in those cases which are essentially gouty or rheumatic, and in these cases laryngeal spasm may prove a grave discomfort, especially during sleep, the patient starting up with an alarming sense of dyspnoea. Women are more prone to severe attacks when menstruating. The affected area may be restricted to certain regions in the larynx, and for such local variations in distribution the names *epiglottitis*, *arytaenoiditis*, or *chorditis* have been employed. Again, if the inflammatory thickening be mainly subglottic, it is described as *laryngitis hypoglottica*, or if haemorrhagic, as *laryngitis haemorrhagica* (see Fig. 3, Plate VII., and laryngeal haemorrhage, p. 194); or *laryngitis herpetica* when vesicles are present. The occurrence of oedema in simple catarrhal laryngitis is extremely rare (Fig. 5, Plate VII.), whilst the presence of well-marked oedema affords strong suspicion of a septic inflammation.

In young children the severity of the symptoms is greatly increased, first, because the glottic aperture is so much smaller, and secondly, because the lymphatic supply of the mucosa is relatively more abundant, and the mucous membrane is more vascular, and less firmly adherent to the underlying structures. Hence the tendency to subglottic infiltration is greater, and this may prove fatal. Moreover, children are more liable to reflex nerve phenomena, such as glottic spasm, and the periodicity in the severity of the symptoms largely depends on this neural factor.

Spasmodic laryngitis (laryngitis stridulosa, or false croup) is essentially an acute laryngitis in a child with the nervous factor predominating, and accordingly accompanied by glottic spasm. This spasmodic laryngitis must not be confused with laryngismus stridulus, a purely nervous affection unaccompanied by laryngeal inflammation, and distinguished from spasmodic laryngitis by the complete intermission of all laryngeal symptoms in the intervals between the attacks. The laryngeal spasm of the affection now under consideration is reflex, and due to the peripheral inflammatory irritation in the larynx, whereas the spasm in laryngismus stridulus is due to central nervous instability (*vide* p. 264).

The usual history is that the child has the symptoms of coryza with some hoarseness, and after going to sleep, awakes suddenly with a severe loud brassy cough and laryngeal spasm. Respiration may be greatly embarrassed and accompanied by inspiratory stridor. After a time the child falls asleep, though remaining restless and disturbed by a croupy cough. The attacks may recur. Towards the morning the symptoms tend to subside, and during the following day the child, with the remission of other symptoms, appears to have lost all tendency to spasm. The following night, however, or the next, the spasmodic attacks recur, but almost invariably with diminished severity.

Treatment.—It is desirable to keep the patient in bed, or at any rate in a room of the temperature of about 64° F., and a steam kettle to moisten the air is usually advisable in severer cases. Abstention from using the voice is important, the bowels should be freely moved, and the diet light. Discomfort may be lessened by inhalations of compound tincture of benzoin, one fluid dram to a pint of water at 140° F. and the steam inhaled, and to this three minims of chloroform may be added if the pain is considerable. Local applications in the form of a spray—for example, menthol gr. vi to viii; cocaine gr. iii to iv; eucalyptol ♏ vi to x to half a fluid ounce of liquid paraffin, or very weak suprarenal gland extract in 4 per cent novocaine hydrochloride aqueous solution—often prove useful. Externally a mustard leaf or a mustard and linseed poultice may be applied to the laryngeal region, whilst small pieces of ice sucked slowly, or cold compresses frequently changed, are often of service. Internally a cough linctus, containing morphine and apomorphine, codeine, or heroin, or small doses of belladonna, with ipecacuanha, may be commended, and tendency to spasm may be combated by chloral, bromides, or phenazone. Pastilles of morphine, menthol, benzoic acid, cubebs, rhatany, or morphine and ipecacuanha lozenges are useful in less acute cases. As the condition becomes subacute more stimulating inhalations are often desirable, such as ʒi oil of Scotch pine or of the pinus pumilio to a pint of nearly boiling water. At this stage a fine laryngeal spray carefully applied, or painting the larynx with solutions of chloride of zinc or nitrate of silver, 10 to 20 grains to the ounce, may be used once or twice, and a nerve tonic administered, such as strychnine, iron, phosphorus, and arsenic combined in the form of a pill.

For the acute attacks of children at night an emetic of ipecacuanha wine or apomorphine often gives more comfort and expeditious relief than anything else. If spasm be marked, $\frac{1}{300}$ to $\frac{1}{200}$ gr. of trinitrin, or chloral and bromide of potassium are of value; the latter may be given per rectum if the child is refractory. When laryngeal obstruction is sufficient to cause grave dyspnoea, tracheotomy or intubation may be required; and in these cases intubation is obviously preferable (if assistance is at hand, in case the tube is coughed out), as the dyspnoea does not last long (*vide* p. 224).

When vocal weakness or aphonia persists after the attack has passed away, it is usually due to paresis of the internal thyro-arytaenoid muscles, and if this does not disappear with the administration of strychnine, local faradisation is very helpful. Singers and professional voice-users should exercise the greatest judgment and patience in avoiding vocal efforts. The too early use of the voice is a prolific cause of more permanent vocal breakdown.

As prophylactic measures, and after recovery from acute symptoms, abundance of fresh air, avoidance of over-heated and ill-ventilated rooms, cold bathing, and general hygienic measures are called for.

Chronic laryngitis very often supervenes on acute attacks, hence all the exciting and disposing causes of acute laryngitis apply equally to chronic laryngitis. But perhaps even more frequently, owing to the persistence of less active causal factors, chronic laryngitis comes on gradually without any acute phase. Thus, prolonged excessive or faulty use of the voice, particularly when associated with other causes, such as alcoholic intemperance, excessive smoking, chronic dyspepsia, gouty conditions, breathing in dusty atmospheres, nasal obstruction with resulting buccal respiration, exposes the pharynx and larynx to unfiltered, unwarmed, and unmoistened air during each inspiration, and is in itself an undoubted cause of chronic or of acute laryngitis, although notable nasal obstruction often exists without such results in the larynx. Suppurative disease of the nasal passages or nasal accessory sinuses is apt to cause laryngitis.

The chief *symptoms* are vocal weakness and huskiness in varying degree, the voice being quickly tired, with a tendency to loss of tone control. As a rule the hoarseness is most marked in the morning, the patient gaining a certain amount of power after using the voice for a time. In the evening, however, the voice again is apt to become hoarser and weaker, and often quite aphonic: a sense of irritation in the laryngo-tracheal region, often compared to the tickling of a hair or crumb in the throat, causes frequent, hawking, irritable cough, which fails to clear the throat. Expectoration of small pellets of mucus is common.

Laryngoscopically the appearances differ considerably with the degree and distribution of the resulting thickening or atrophy of the mucous membrane and submucous tissues. The following clinical types are recognised: (*a*) simple chronic laryngitis, without much alteration in the character or contour of the mucosa, but with localised reddening of the vocal cords and vocal processes, and increased secretion, which is apt to form sticky bands stretching across the glottic aperture; (*b*) hypertrophic laryngitis, in which there is marked hyperplasia of the mucous membrane, either local or general; the ventricular bands are sometimes so greatly thickened that they overlap and partially or completely obscure the true cords. Hyperplastic changes limited to the cords, particularly to the region of the vocal processes or to the inter-arytaenoid fold, are known as pachydermia laryngis (Fig. 1, Plate IX.), and constitute a separate clinical group of sufficient importance to require fuller description in connexion with benign laryngeal neoplasms (see p. 239). In the subglottic form of hyperplasia, known as *chorditis vocalis hypertrophica inferior*, an infraglottic swelling is seen below the vocal cords on deep inspiration. Many of these cases, however, are of infectious origin, and belong to the class described as scleroma (see p. 140); (*c*) atrophic laryngitis, in which the mucous membrane is shrunken and atrophic, often associated with atrophic conditions of the nasal mucosa. Here, especially, the scanty secretion is prone to be sticky and even to form crusts. Sometimes there are firmly adherent green crusts on the vocal cords, and these may be so extensive as to cause serious dyspnoea. The absence of normal fluid glandular secretion may cause much irritation and discomfort, and the term *laryngitis sicca* is applicable to such conditions; (*d*) granular or glandular laryngitis, in which the enlarged racemose glands are seen standing out from the generally shrunken surface of the mucous membrane, whilst not rarely the cords themselves may look granular; (*e*) oedematous laryngitis is sometimes seen in chronic cases, though more commonly in the acute form. It is characterised by diffuse or localised oedematous infiltration (Fig. 5, Plate VII.).

Differential Diagnosis.—It is necessary to exercise caution in the diagnosis of subacute or chronic laryngitis, because the symptoms and laryngoscopic appearances may be closely simulated by, or may be indistinguishable from, those of more serious conditions, of which the chronic laryngitis is a secondary manifestation only. The greatest difficulty will be encountered in excluding tuberculosis; but secondary syphilitic laryngitis, or even an early diffuse malignant growth, may also afford much difficulty in recognition. Suspicion should always be aroused with regard to the possibility of graver mischief underlying the appearances when the laryngeal inflammation is restricted to one side, especially when only one vocal cord or ventricular band is affected. The fact that the epiglottis alone or the inter-arytaenoid fold is the only implicated region points strongly to some infective process. The differential diagnosis of these diseases is discussed at greater length in the section on malignant disease of the larynx (p. 245). But it will suffice here to emphasise the necessity for caution in too readily assuming that an apparently innocent laryngitis is not in reality a far graver affection, for hoarseness is commonly the only early symptom that attracts the notice of the patient in early intrinsic laryngeal cancer, and is often the chief complaint in early tuberculosis or syphilitic disease, and the ready-made diagnosis of the patient, namely, laryngitis following a cold, is at times accepted by the medical attendant without due investigation, until perchance the period for successful interference has passed for ever.

Certain laryngeal neuroses form a totally different group of affections, which must be mentioned as simulating laryngitis, because their dominant symptom is hoarseness or loss of voice. Perhaps the least likely to present difficulties is functional aphonia, and yet when it is said to have been ushered in by a cold, or when its origin actually dates from a coryza with laryngitis, its truly neurotic basis may be mistaken for muscular paresis of the thyro-arytaenoidei interni. From the latter it can be differentiated by the rule that in organic paralyses the cough is aphonic, whilst of course the general condition of a neurotic female in itself points strongly towards the diagnosis of functional aphonia. More important, yet more frequently unrecognised, are the organic paralyses, such as complete paralysis of one vocal cord from pressure on the corresponding recurrent laryngeal nerve by an intrathoracic aneurysm or growth, or an enlarged thyroid gland, and so forth, or paresis of the internal tensors in early tabes dorsalis, or bulbar paralysis. Needless to say, ankylosis of the crico-arytaenoid joint too may be overlooked when the thickening round the joint is very slightly marked.

Treatment.—The recognition of any disposing affection naturally leads to appropriate measures being adopted for their removal, such as anti-rheumatic medication, treatment of gout, dyspepsia, anaemia. Abuse of tobacco and alcohol must be strictly prohibited. Buccal respiration from nasal obstruction calls for treatment directed to the relief of nasal abnormalities. In professional voice-users, improper vocal production may have to be corrected. School teachers are prone to suffer from constant speaking in dusty atmospheres, particularly from the chalk dust of a dry blackboard-duster. Vocal failure in singing is often due to faulty methods of breathing, wrong use of the vocal registers, and over-use of the voice when suffering from catarrh. The necessity of vocal rest cannot be too strongly insisted upon in professional voice-users suffering from chronic laryngitis. Failure of any form of treatment is almost certain in such cases, unless accompanied by vocal rest until the chronic inflammation has subsided.

In a good many patients, particularly in plethoric persons, the general treatment is of paramount importance, and in all cases too much stress can hardly be laid on attention to general health, and, in addition to cold bathing, the administration of iron, quinine, strychnine, or phosphorus in various combinations is of very great value. In obstinate cases, a course of treatment for three or four weeks at some spa, such as Ems, Mont Dore, Cauterets, Marienbad, Kissingen, or Aix-les-Bains, or in our own country at Harrogate, particularly if combined with baths and massage, may prove very useful, followed if necessary by a fortnight's stay in some bracing climate.

Locally stimulating inhalations of oil of Scotch pine (a dram to the pint of water at 140° F.) or creasote, or turpentine, or a fine intra-laryngeal spray of some silver preparation, such as argyrol or protargol 10 per cent solution in water, or even nitrate of silver gr. v-xx to the ounce of water, or chloride of zinc gr. x to xx to the fluid ounce, are required in inveterate cases. In such cases electric massage of the neck is also of great service.

Of internal remedies salicylate of sodium gr. v to x, or iodide of potassium in small doses thrice daily, has proved very useful in the experience of one of us (W. W.). Chloride of ammonium and cubebs may be tried in the same way. Pastilles containing cubebs, benzoic acid, guaiacum resin, or Soden mineral pastilles are of use in many cases.

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OEDEMA OF THE LARYNX.—Though oedema of the larynx does not represent a distinct disease, but a complication associated with various morbid states either local or general, yet on account of the importance of many affections in which oedema is the dominant manifestation, and for the sake of obtaining a comprehensive view of its clinical features, it is desirable to devote a separate section to this subject.

We recognise three clinical groups of laryngeal oedema: (1) simple inflammatory oedema, (*a*) primary, (*b*) secondary; (2) non-inflammatory or passive oedema; (3) acute septic inflammatory oedema (*vide* p. 117).

Acute Inflammatory Oedema.—Non-infective inflammatory oedema may be due to trauma from swallowing very hot fluids, from the impaction of some hard or pointed body, or from some irritant inhaled, swallowed, or applied to the larynx. Inflammatory oedema very rarely supervenes on acute catarrhal laryngitis. It may arise in the course of scarlet fever, enteric fever, typhus fever, small-pox, and influenza, and has occurred in hydrophobia. Secondarily it occurs in syphilitic or tuberculous disease of the larynx, or as the result of cancerous infection, or by extension of suppuration in a deep cervical gland. A very large proportion of cases of laryngeal oedema are in this sense secondary, especially to infective disease setting up perichondritis (Fig. 6, Plate VII.).

Laryngoscopically the parts most frequently implicated are the epiglottis, the ary-epiglottic folds, the ventricular bands, and the subglottic region on account of the lax nature and richness in lymphatic vessels of the submucous tissues, whereas the vocal cords to which the mucous membrane is firmly adherent are more rarely the seat of oedema. The exudation is usually serous, but may be blood-stained, and the affected regions are bright red or greyish-pink, and swollen, with a tumid, rounded, tense aspect (Fig. 5, Plate VII.).

The *symptoms* are those of acute catarrhal laryngitis, but with more pain, dyspnoea, and greater constitutional disturbance. When the ventricular bands or the subglottic submucosa are much swollen, dyspnoea may be marked, whilst inflammation of the epiglottis is more liable to cause pain in deglutition.

The *treatment* of these conditions is much the same as that of the severer forms of acute catarrhal laryngitis. In all cases absolute rest in bed, in a warm moist atmosphere, is essential, and complete abstention from speaking should be enjoined. But in the more marked cases the continuous application of cold externally in the form of ice-packs, or Leiter's tubes, afford relief, or leeches may be applied. Free scarification of the oedematous swelling, after local anaesthetising, may be resorted to if dyspnoea is threatening, but should not be done unless other means have failed or unless the symptoms are urgent. The danger of rapidly increasing dyspnoea and consequent asphyxia must always be before the medical attendant, and provision made for its relief by tracheotomy or intubation at short notice. The advantage of intubation in skilled hands is that no wound is made in the inflamed area, whilst the compression of the tube tends to cause absorption of the exudation; on the other hand, the oedematous swelling may make it impossible to introduce a full-sized tube, and the smaller one may soon become loose and be coughed out. Internally diaphoretics may be given, and to overcome any tendency to spasm bromide of potassium in 10 to 20 grain doses. The food should be cool or cold, bland, and non-irritating, and if deglutition be very painful the desirability of rectal alimentation may arise.

Non-Inflammatory or Passive Oedema.—*Etiology.*—The causes are general as distinguished from the local causes of inflammatory oedema. They are those capable of giving rise to general dropsy, such as renal disease, certain valvular diseases of the heart, disturbances of collateral circulation, such as arise from pressure on the veins of the neck by tumours; certain blood conditions, for example, angioneurotic oedema, or oedema from the use of iodide of potassium. It sometimes supervenes in syphilitic or cancerous affections of the larynx, and more rarely in tuberculous laryngitis and in diphtheria.

The *symptoms* are mainly mechanical, and are either those of laryngeal obstruction or interference with the action of the vocal cords in using the voice. Objectively the affected regions are swollen, translucent to greyish or greyish-pink in colour.

The *treatment* called for is mainly the relief of dyspnoea when present, which differs in no respect from that of the inflammatory form. When it is due to cardiac or renal disease, general measures appropriate to these affections should be employed. Strübing recommends ice and morphine for angioneurotic oedema, or small doses of atropine or pilocarpine may be tried. For oedema due to iodide of potassium the free administration of bicarbonate of sodium has proved successful.

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Laryngeal Haemorrhage.—Extravasation of blood from the laryngeal vessels may be submucous, or it may be poured out on the surface, and hence we divide such cases into two groups: internal or submucous haemorrhage and external haemorrhage. When the blood remains submucous it may form a firm clot or become organised; it may then simulate in its symptoms, and sometimes in laryngoscopic appearances, a neoplasm if defined in area, or some infiltrating infective disease if more diffused. Small submucous haemorrhage on a vocal cord may imitate a diffuse malignant infiltration, or may be so limited in extent as to be readily recognisable as a simple blood extravasation, somewhat like subjunctival haemorrhage. When the extravasated blood is localised and forms a definite tumour it may be hardly distinguishable from a true neoplasm; thus, one of us (F. S.) has described cases which resembled an angioma, cancer of the vocal cord, and a soft fibroma (see Fig. 8, Plate X.) respectively.

External laryngeal haemorrhage is characterised by the expectoration of blood. In acute laryngitis it is not unusual for the sputum to be streaked with blood, and sometimes considerable amounts of blood may be lost; this has occurred especially in influenza. As a rule the symptoms are too slight to call for special note, but when the blood is poured out in large quantities, the blood-clots by blocking the glottis may cause great dyspnoea till they are expelled. Such blood-clots may be retained for a considerable time, and laryngoscopically resemble neoplasms. It is well to remember that blood which is coughed up probably comes from the lungs, unless on laryngoscopic examination the actual source of bleeding in the larynx can be observed. And when it does come from the larynx, unless there is some syphilitic, malignant, or tuberculous ulcer, or is due to some traumatic cause, such as external or internal injury or excessive and violent use of the voice or a foreign body, there is generally some underlying cause, such as purpura, leukaemia, chlorosis, renal disease, or general arterial degeneration.

The *treatment* consists in the measures appropriate to any underlying or disposing cause and the removal of any exciting cause, if possible. For the arrest of bleeding, rest, the application of cold, sucking ice, or a laryngeal spray of weak adrenalin solution are suitable. Blood-clots, whether external or submucous, should be removed if they cause troublesome symptoms. Slighter submucous haemorrhage may be left to be absorbed.

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Tuberculosis.—*Causes.*—Laryngeal tuberculosis is one of the most frequent complications of the same disease in the lungs, and, according to Heinze's pathological investigations, is met with in about 30 per cent of all cases of pulmonary phthisis, whilst, according to general experience, the percentage is about 20 per cent. It is commonly supposed that the laryngeal complications of pulmonary tuberculosis usually occur in the later stages of the disease; the recent experience of one of us (F. S.) at King Edward VII. Sanatorium, however, has shewn that laryngeal tuberculosis occurs in an unexpectedly large number of cases of early pulmonary phthisis. The occurrence of primary laryngeal tuberculosis is now definitely established by the results of a few post-mortem examinations, but it is an event of the greatest rarity. It is much more frequent in men than in women, and its more severe forms also occur more often in the male sex. It is seen at all ages, but occurs most frequently in the years of early manhood. From the diagnostic point of view it is important to remember that senile laryngeal tuberculosis is by no means rare. Disregard of this has only too often led to deplorable diagnostic mistakes. The determining cause of the disease is the *Bacillus tuberculosis*; constitutional weakness and unfavourable conditions of life play the part of disposing factors. What determines the occurrence of the laryngeal complication is not yet certain. The disease may begin on the surface and penetrate into the deeper tissues, or start in the opposite direction; the former probably is the more frequent contingency.

Pathology.—The deposit of tubercles in the larynx is usually manifested by infiltration and pseudo-oedematous thickening of the tissues. This is most marked, as a rule, in the epiglottis, the arytaeno-epiglottidean folds, the mucous membrane covering the arytaenoid cartilages, and the inter-arytaenoid fold. In another series of cases, however, the disease begins on the vocal cords or on the ventricular bands in the shape of swelling or of superficial ulceration; no part of the larynx is immune against the invasion of tubercle. The stage of actual infiltration is often preceded by marked anaemia of the mucous membrane of the whole larynx, usually associated with an analogous condition of the pharyngeal mucous membrane; this anaemia is most noticeable on the epiglottis. In some cases the contrast between the healthy appearance of the patient's complexion and the pallor of the larynx is most striking. In very rare cases tubercles themselves have been seen as small yellowish or greyish nodules in the midst of the general infiltration; the stage of their corpuscular existence, however, must be extremely brief, and in the great majority of cases the first sign of their presence is manifested by the small superficial ulcerations which result from their disintegration. These ulcers quickly coalesce, extend in width and depth, and after a time give a worm-eaten appearance to the parts attacked. The epithelium, the mucosa, and submucosa having been destroyed, they extend towards the perichondrium and lead to perichondritis, caries, necrosis, and sometimes to exfoliation of parts of the cartilages. Actual tuberculous tumours, consisting of an aggregation of miliary tubercles and cellular infiltration of the mucosa and submucosa, as well as of general debris, are met with in any part of the larynx, and this even in cases in which there is no evidence of concomitant lung disease. Their occurrence is not so widely known as it deserves to be, in view of the possibility that they may be mistaken for benign or malignant laryngeal growths. It certainly is more frequent than might seem from the small number of published cases.

Symptoms.—The subjective symptoms of laryngeal tuberculosis are, according to the seat of the disease, either hoarseness, and, in later stages, more or less complete aphonia, or pain, difficulty in swallowing, cough with more or less expectoration, and sometimes dyspnoea. Hoarseness and aphonia will be met with when the vocal cords or ventricular bands are implicated; difficulty in swallowing when the epiglottis, the posterior wall of the larynx, and the ary-epiglottic folds are the parts concerned; pain, whenever fibres of the superior laryngeal nerve are laid bare or otherwise affected. Often all these symptoms are met with simultaneously. The most troublesome of these are usually cough, pain, and dysphagia. Whilst all the symptoms named depend, as a rule, upon the local conditions, the cough may also, of course, be due to the concomitant pulmonary disease; moreover, the vocal troubles are not necessarily due to the swollen and ulcerated state of the vocal cords, but may depend upon implication of the right recurrent laryngeal nerve in pleuritic thickening at the apex of the right lung, or upon pressure of enlarged bronchial glands upon one or both recurrent laryngeal nerves, and subsequent paralysis of the corresponding vocal cord or cords; the shortness of breath often observed in these patients is more commonly due to the concomitant pulmonary affection than to the laryngeal trouble; though

PLATE VIII

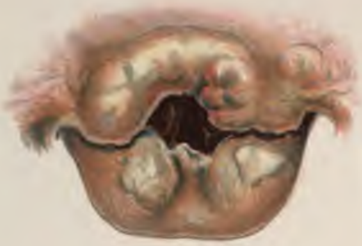


Fig. 1.

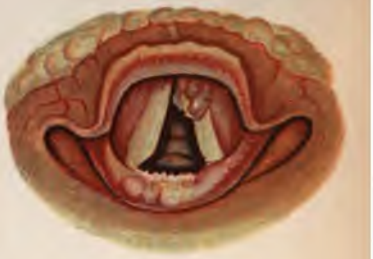
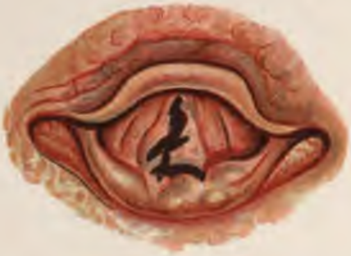


Fig. 2.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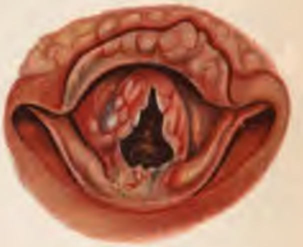
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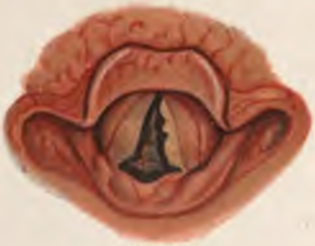
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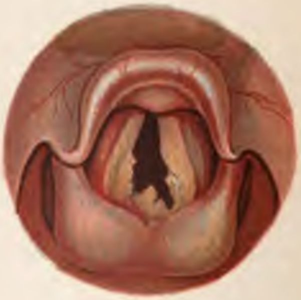
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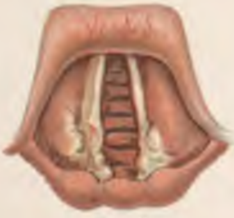
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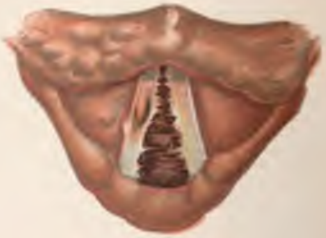


Fig. 8.

PLATE VIII.

LARYNGEAL TUBERCULOSIS AND LUPUS.

Fig. 1. Tuberculosis. The epiglottis is breaking down, and extensive ulceration has occurred. The arytaenoid regions shew typical pear-shaped infiltration. (P. W. W.)

Figs. 2, 3, 4, and 5. Laryngeal tuberculosis. (Smurthwaite.)

Fig. 6. Tuberculosis with ulceration of the vocal cord, and a tuberculous pachydermia in the inter-arytaenoid fold. (Krieg.)

,, 7. Superficial tuberculous ulceration of the ventricular bands, and of the right vocal cord. The general pallor of the mucous membrane is very noticeable. (P. W. W.)

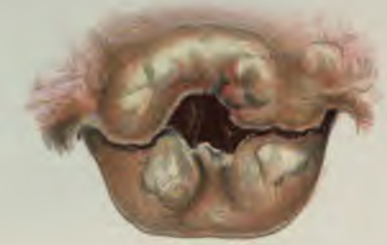
,, 8. Lupus of the larynx. (P. W. W.)

in later stages it may be of laryngeal origin, taking its rise either in general oedematous swelling of the larynx or in implication of the crico-arytaenoid joints, with diminished mobility or immobility of the vocal cords as the result of perichondritis. In very rare cases bilateral paralysis of the abductors of the vocal cords, due to pressure of enlarged bronchial glands upon the recurrent laryngeal nerves, may produce the same effect.

Objectively, the pallor of the mucous membrane, preceding any definite signs of actual tuberculous mischief, and persisting generally throughout all subsequent stages, is of diagnostic value (Fig. 7, Plate VIII.). When met with in any case in which there is not general anaemia the patient's lungs must be minutely examined. On the other hand not rarely the initial symptom may be obstinate laryngeal congestion, which, when concerning both vocal cords, at first is indistinguishable from ordinary laryngeal catarrh. Congestion confined to one vocal cord should always remind the observer that this may be the danger signal of graver constitutional mischief (tuberculosis, syphilis, malignant disease). Undoubtedly, however, cases of unilateral congestion occur, in which this symptom is not the forerunner of more grave disturbances, and no alarmist apprehensions ought to be expressed on the strength of this sign alone.

When tuberculous infiltration takes place, and particularly when this pre-eminently concerns the epiglottis and the mucous membrane over the arytaenoid cartilages, as in many cases it does, the appearances often are so characteristic as to enable an experienced observer to diagnose the existence of tuberculosis with tolerable certainty, independently of the condition of the lungs, which, however, needless to say, must never be neglected. In such cases the epiglottis is changed into a pale, rounded sausage- or turban-like body, several times its normal size, lying across the pharynx; thus inspection of the interior of the larynx proper is often prevented, whilst the arytaenoid cartilages are changed into two puffy, pale, rounded or pyriform bodies, which, together with the epiglottis, completely fill up the image seen in the laryngeal mirror. The oedema is distinguished from ordinary oedema by its greater density. Later, the surface of these swellings, originally smooth and shiny, becomes completely riddled with small superficial ulcers, which quickly coalesce and give to all the parts affected the worm-eaten appearance already described. In other cases tumefaction first begins in the inter-arytaenoid fold; and when ulceration occurs, small stalactite-like projections may be seen in that part (Fig. 6, Plate VIII.). Again, in a third class of cases the infiltration and ulceration may begin on one or both vocal cords or ventricular bands, and sometimes the only laryngeal manifestation of tuberculous disease of that part consists in complete erosion of one or both the cords by ulceration. In later stages the whole laryngeal mucous membrane often forms one mass of ulceration, which does not remain superficial, but gradually spreads towards the submucosa, the perichondrium and the cartilages themselves. The epiglottis may be destroyed in part or entirely; sometimes indeed a

PLATE VIII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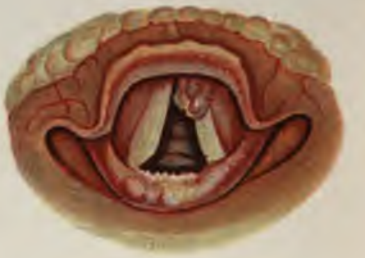
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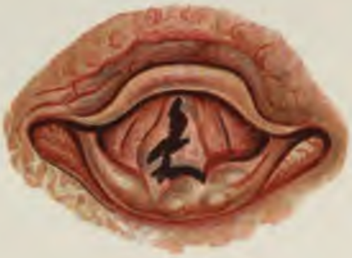
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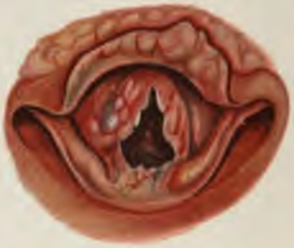
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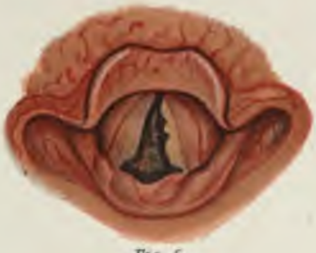
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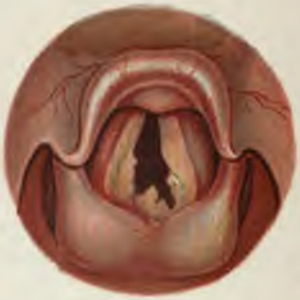
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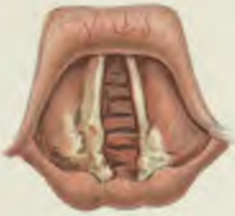
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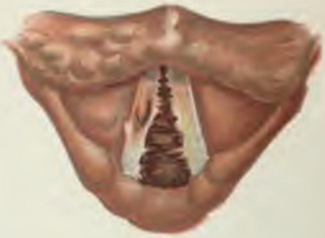
Fig. 8.

present as it was in former days. If the pulmonary lesions be still limited to consolidation of the apices, and if the laryngeal ulcerations be not too extensive and situated in the interior of the larynx, it is now possible by a judicious combination of constitutional and local treatment to arrest the disease in not a few cases; although, of course, even when we have succeeded in bringing about cicatrisation of a tuberculous ulcer, we must always be prepared for fresh manifestations.

Treatment.—In cases in which both the pulmonary and the laryngeal lesions come under observation, when they are at an early stage we strongly recommend a combination of sanatorium treatment with absolute and prolonged silence, which affords the larynx complete rest. Moritz, Schmidt, and Lublinski in Germany, and Dr. St. Clair Thomson in this country, were amongst the first to emphasise the value and importance of vocal rest in the treatment of laryngeal tuberculosis. As the experience of one of us (F. S.), of Drs. Bardswell and Adams at King Edward VII.'s Sanatorium, as well as of one of us (W. W.) at other sanatoria, and Dr. Felkin's observations at the Ringwood Sanatorium shew, often very satisfactory, sometimes truly surprising results are obtained, even in more advanced cases, when the patients can be brought to see the value of the method and adhere rigidly to the undoubtedly very irksome and depressing régime. We wish, however, expressly to state that the plan is not infallibly successful, and is in no way intended to supersede, in cases in which this is really required, local treatment, with which, on the contrary, it may be most usefully combined. We also beg to protest against its adoption in cases in which the existence of laryngeal tuberculosis is merely suspected, not proven. A warning to this effect is, to conclude from recent personal experiences, by no means superfluous. The length of time during which absolute silence is required, of course, greatly varies in individual cases. To ensure success the moral support of the superintending physician is of the greatest importance. Except in very rare cases the method is not suitable for home treatment. Locally, when the ulceration is limited, we employ, after previous cocainisation, applications of pure lactic acid. The drug is firmly rubbed into the ulcerated parts by means of Krause's forceps, round which a small pellet of cotton wool is securely wound. The practitioner should remember that these applications are not to be made in the gentle fashion of an ordinary astringent application, but in that of the cleansing of a tuberculous joint. If the ulceration be at all deep, the application of the lactic acid must be preceded by scraping the base of the ulcers by means of Heryng's curette, exactly as one would scrape the granulating surfaces of a tuberculous joint after it had been opened. This means, however, should be practised only by operators fully conversant with more delicate intra-laryngeal operations, for by an indiscriminate use of the curette more harm than good may be done. In not a few cases the results of this treatment, when properly carried out, are most gratifying. Should the ulceration be too far advanced, and the general condition of the patient be at too low an ebb to admit of energetic treatment, local sedatives—such as insufflation of anaesthesine or orthoform before meals—should be employed. The use of these powders will also be found very useful in cases of distressing laryngeal cough and especially of dysphagia. Their effect lasts much longer than that of cocaine, eucaine, or morphine, and the dose need not be increased so rapidly as that of the last-named drugs, whilst, in our experience, they are perfectly innocuous. Should the difficulty in swallowing be due to infiltration and ulceration of the epiglottis, relief will often follow if the patient takes fluid nourishment in the horizontal position, the nozzle of a feeding-cup being introduced in the dependent angle of the mouth. In many such cases amputation of the epiglottis does away entirely with the distressing dysphagia. Grünwald has recently warmly recommended deep galvano-cauterisation of infiltrated parts, and his recommendation has been seconded by Besold and Gidionsen. Tracheotomy will be required very rarely. Our own results in cases of laryngeal tuberculosis so far have not been particularly encouraging, and it should be remembered, when this operation is under consideration, first, that in a weak patient with large cavities in his lungs, coughing and expectoration will be rendered more difficult when an opening is made in the trachea; and, secondly, that there is a very real danger of tuberculous infection of the wound. The last objection, of course, applies also with regard to thyrotomy, which otherwise has been successful in a few cases. One of us (W. W.) has obtained good results from submucous injection of guaiacol in almond oil, where the tuberculous deposit is localised and the overlying mucosa intact. The local reaction is usually attended by marked relief to pain, and in favourable cases repeated injections have resulted in the disappearance of the disease. Total extirpation of the larynx will hardly ever be seriously considered, seeing that we have to deal with a general, not a purely local malady. With regard to constitutional treatment we consider the employment of tuberculin injections under the control of the opsonic index as still entirely *sub judice*. In cases in which the laryngeal or pulmonary disease, or both, are too advanced to allow of their admission into a sanatorium, or in which sanatorium treatment is for other reasons impossible, we still give pure creasote in gelatin capsules, each capsule containing one minim of the drug. The patient begins by taking one capsule three times daily immediately after meals, and increases the dose gradually from one to five capsules three times daily.

General and hygienic treatment must of course go hand-in-hand with such local measures as may be required. To avoid repetition we refer the reader for details as to the general management of these cases to the article on pulmonary tuberculosis in Vol. V. of this work. In conclusion, we only wish to say that if a change of air should be considered advisable, either a sea-voyage or a stay at Bournemouth, or Torquay, or any of the health resorts of the Riviera, of Southern Italy, or of the North of Africa may be advantageous. In accord with the experience of most practitioners in the Engadine, we have found that the existence of laryngeal complications is a serious drawback to residence at those high altitudes, however desirable it may be from the point of view of the pulmonary disease.

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Lupus of the Pharynx and Larynx.—*Causes.*—The nose and throat are involved to a greater or less extent in a large percentage of cases of cutaneous lupus; in a small proportion, however, this disease originates and may long exist in the pharynx and larynx, without the external integument or the nose becoming affected.

As regards the etiology of the affection, it is directly due, no doubt, to a specific bacillus; and the great majority of writers are agreed in regarding lupus and tuberculosis as one and the same disease under different conditions. But whilst the identity of the specific microbe of lupus and tuberculosis is generally, though not universally, admitted, the cause of the remarkable difference in the clinical condition seen in these diseases, especially as it manifests itself in the mucous membrane of the upper air-passages, has yet to be explained.

The disease generally reveals itself between the ages of two and ten years. It is more liable to occur in women than in men, and arises more frequently in persons of an inherited tuberculous proclivity, though lupus patients are themselves but rarely the subjects of ordinary tuberculous disease; nor does the particular affection itself shew any marked tendency to hereditary transmission. The disease is in no way connected with syphilis, although frequently in its physical aspects it is hardly distinguishable from the lesions of syphilis.

The nodules and tumefaction which are characteristic of lupus consist of a cellular new growth in the mucous membrane in which giant cells, and occasionally bacilli indistinguishable from tubercle bacilli, may be found. When the deposit first manifests itself on the uvula or on the free border of the soft palate, we may find localised tumefaction, generally of distinctly heightened colour, less marked and more limited than in syphilis or acute pharyngitis, but differing in aspect from the anaemia premonitory of tuberculosis; sometimes the deposit appears in mucous membrane apparently healthy. In course of time, smooth, hard nodules appear, varying in size from a pin-head to a split-pea, and generally of a rosy pink colour.

The nodular deposit greatly deforms the parts; and when arising in the uvula or soft palate the distortion and twisted appearance of the affected structures may be well marked.

Soon the nodules become softer and characteristically "apple-jelly-like" in appearance, and then as a rule ulceration begins. The ulcers present a serpiginous worm-eaten appearance, with defined hard or soft, granular and prominent margins, and a velvety, red, dry, indolent base. The process of ulceration progresses very slowly, healing in one direction while spreading in another; and periods of increased activity alternate with long periods during which the disease appears to remain in abeyance.

When the tonsils are involved, they become covered with irregular red nodules and pits of ulceration, but the course of the disease is precisely similar to the faucial deposits.

In the larynx, lupus generally attacks first the free margin of the epiglottis, which becomes tumefied; and the swelling gradually spreads to the arytaeno-epiglottic fold and ventricular bands. The epiglottis becomes pale, "worm-eaten," and rough in aspect, and large portions may be completely lost. The vocal cords are usually the last part to be affected, and so slow is the progress of the disease that they often escape. When attacked, they become red and tumefied (Fig. 8, Plate VIII.).

Symptoms.—As pain is entirely absent or very slight in lupus, the pharynx and larynx are often invaded without any obvious symptoms until the destruction of the soft palate causes fluids to return through the nose on swallowing, or gives a nasal tone to the voice and renders articulation imperfect. From the invasion of the posterior commissure or the vocal cords the voice becomes hoarse and aphonic. Some degree of stiffness in the pharynx and slight soreness and tickling sensations may be felt in the pharyngo-laryngeal region. In the advanced laryngeal disease stenosis and dyspnoea very often arise and may necessitate tracheotomy, but there is hardly ever any inflammatory exudation with acute dyspnoea; the laryngeal stenosis is always very slowly established, and ample warning is given of the increasing urgency of the dyspnoea. Active perichondritis or necrosis of cartilages is excessively rare.

Diagnosis.—If there should be coexistence of cutaneous lupus with manifestations of the disease in the pharynx and larynx, there will hardly ever be any doubt as to the correct interpretation of the pharyngeal and laryngeal phenomena. On the other hand there may be very considerable difficulties, if the pharynx or larynx is affected primarily; the differential diagnosis has then to be made from simple chronic pharyngitis or laryngitis, syphilis, tuberculosis, and carcinoma.

Chronic pharyngitis is attended with increased secretion, and the hypertrophied lymphatic follicles are confined to the posterior and lateral walls, whilst there is no distortion of the parts, and the absence of lupus tubercles is to be noted. In the earlier stages of syphilitic laryngitis, especially in the catarrhal form with or without superficial ulceration, in the later stages with diffuse infiltration, and more especially in hereditary cases, the laryngoscopic appearance and the age of the patient often fail to settle the diagnosis till antisyphilitic remedies have been tried. Yet even at first the aspect of the tumefaction, the distortion of the parts, and the slow erosion of the tissues without distinct and obvious ulceration, are generally enough to lead, at any rate, to a strong suspicion of the real nature of the affection. The rule that lupus usually occurs in the very young, is very slowly progressive, always with cicatrisation, and is hardly ever painful, together with the peculiar appearances of the growth, and the absence of wasting, fever, or quickened pulse, should rarely leave any doubt as to the differential diagnosis from ordinary tuberculosis, which is characterised by general pallor of the mucous membrane, and numerous mouse-nibbled, pale ulcers covered with greyish, disintegrating tuberculous tissue, and is usually accompanied by considerable pain, especially in swallowing.

The prognosis as regards life is favourable, the chief danger being stenosis of the larynx; but this comes on so gradually, and is so little prone to be suddenly increased by perichondritis or oedema, that tracheotomy can almost always be performed in good time. Occasionally long-standing lupus of the pharynx and larynx ends in pulmonary tuberculosis.

In cases in which the disease is confined to the pharynx and larynx, and is accessible, good results may be obtained by vigorous treatment, and occasionally complete cures.

Treatment.—Patients affected with lupus should be placed under the most favourable hygienic conditions possible, and during the winter months should take cod-liver oil; general tonic remedies such as the syrup of iodide of iron or arsenic should be given from time to time.

The remark made in the section on tuberculosis concerning the uncertainty as to the effect of tuberculin injections under the control of the opsonic index applies to lupus of the larynx also.

As regards local treatment, the nodules and tumefactions should be scarified or curetted, and strong lactic acid (80 per cent) rubbed in after the same manner as in tuberculous disease of the larynx. This should be done once a week, successive portions being treated until the whole of the diseased area has become cicatrised and no nodules or ulcers are visible. The cases should be watched for at least a year after apparent cure has been effected; and any fresh manifestations of the disease should be similarly dealt with at once.

Isolated deposits may be destroyed by the galvano-cautery. In a case shewn before the Clinical Society of London by one of us (F. S.) many years ago, lasting and complete cure of a very extensive laryngeal lupus had been obtained by persistent use of this form of cautery.

Stenosis of the larynx may be arrested for a time by intubation or by the passage of Schroetter's bougies; occasionally tracheotomy may be unavoidable.

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Leprosy of the Pharynx and Larynx.—For a general account of this disease the reader is referred to the article "Leprosy" in this work (Vol. II. Part II. p. 648).

Laryngeal Symptoms.—The larynx is especially susceptible to leprosy, but the disease never appears in the larynx primarily. It usually attacks this organ after it has invaded the skin, mouth, and fauces.

Leprosy of the throat may assume the tubercular or, very rarely, the anaesthetic variety. In either case the onset is extremely insidious, owing to the painless nature of the affection; and patients will sometimes declare that they have nothing the matter with the throat when examination reveals undoubted evidence that it has been established there for a long time.

Tubercular leprosy of the mucous membrane passes through three stages. In the first stage the uvula and soft palate, in which the alterations are usually first observed, become red and velvety in appearance, and the neighbouring tissues become affected by continuity or by separate foci of disease, so that the epiglottis and arytaeno-epiglottic folds likewise become red, velvety, thickened, and hard, and appear as though coated with varnish. At this stage epistaxis frequently occurs, and the patient may complain of shortness of breath and a sense of tickling and dryness in the pharynx and larynx. In course of time the red, hard infiltration becomes soft, and the tissues somewhat oedematous, the redness soon giving place to pallor, till the affected regions are uniformly pale and resemble the anaemia of tuberculous disease; and when the infiltration and cellular elements become absorbed, the tissues appear, as Mackenzie puts it, as though infiltrated with tallow.

The second stage begins with the formation of the characteristic tubercles, and with the diminution or disappearance of the swelling and tumidity of the mucosa. At first they appear as small nodules of a whitish-yellow colour, or white and almost glistening, varying in size from a pin's head to a split-pea, isolated or in chains and groups, and sometimes surrounded by a hyperaemic areola. In this condition they may remain stationary for years, till the third stage is reached, provided the patient does not succumb to the general affection in the meanwhile. In this stage ulceration and disintegration of the tubercles take place. The ulcers at first are small and rounded, are elevated above the surrounding mucous membrane, and are compared by De la Sota to syphilitic mucous patches. Eventually they become deeper. The glottis assumes a rounded form, and the voice is lost. The fetor of the breath at this stage becomes unbearable. The cartilages of the larynx become involved, the epiglottis presents a knobby aspect and may become hard and distorted, and in course of time the cartilages become necrosed and exfoliated.

The earliest indication of the throat affection consists in alteration of the quality of the voice, which at first becomes nasal, and with the implication of the larynx may be thick; yet the larynx may be extensively diseased without attracting the notice of the patient. Hoarseness or aphonia appears later from implication of the vocal cords. Dyspnoea sometimes supervenes; and stenosis of the larynx, produced either by the nodular infiltration or oedema, may even necessitate tracheotomy.

The anaesthetic variety rarely affects the throat, and, according to Hillis of Demerara, it never does so until the cutaneous affection has existed for five years. The mucous membrane is smooth, the affected regions become anaesthetic, the velum palati is thin, tense, and paretic, and the arches of the palate assume a violet colour.

Diagnosis.—Leprosy has to be distinguished from syphilis, tuberculosis, lupus, and cancer; though laryngeal leprosy practically never occurs without cutaneous manifestations of the malady, and pharyngeal leprosy very rarely. Moreover, a leprous patient may be affected also with cancer, lupus, syphilis, or tuberculosis; or, on the other hand, patients suffering from any one of these diseases may be attacked by leprosy.

Syphilitic throat lesions have much in common with leprosy: first, in that they are usually painless, although the actual anaesthesia of the leprous larynx is not observed in syphilis; secondly, in the hyperaemia of the affected tissues; and, thirdly, in the tendency in the later manifestations of both affections for the cartilages to be attacked.

De la Sota states that the resemblance between syphilitic mucous patches and leprous ulcers is sometimes very close; but the dark reddish or coppery tint produced by syphilis contrasts with the greyish-red that is observed in leprosy. Secondly, the anaesthesia in leprous patches is distinguished from the hyperaesthesia that may attend syphilitic lesions of the perichondrium. Thirdly, syphilitic ulceration does not go beyond a superficial erosion, whilst leprous ulcers, though true ulcers, yet are not so round and deep as tertiary syphilitic ulcers. They are much longer in formation, and do not respond to antisyphilitic treatment; indeed they are often made worse by it. The leprous nodules of the second stage are characteristic.

Tuberculosis in its earlier stages is attended with anaemia and hyperaesthesia of the parts; leprosy gives rise rather to hyperaemic infiltration. Leprosy is sometimes attended with febrile symptoms, but its onset is usually most insidious. The vocal cords are often affected early in tuberculous laryngitis; in leprosy the epiglottis and ventricular bands are generally invaded before the vocal cords. Leprous ulcers are more defined, less irregular than the painful tuberculous ulcers. Lupus may attack the larynx primarily, and De la Sota points out that the absence of cutaneous lesions is therefore a sufficient distinction from leprosy. Lupus arises in a healthy mucous membrane; the leprous tubercles are always preceded by a reddish coloration, which afterwards turns white. Leprous tubercles are white, soft, and variable in size. They appear in the form of a chain or a rosary, and their sensibility may be normal, diminished, or entirely abolished; lupus nodules are of a rosy or reddish hue, hard, resistant, and elastic, larger in size than those of leprosy, and, though indolent, of normal sensibility. Leprous ulcers are superficial, have indistinct edges, and suppurate but little; those of lupus have hard, elevated borders, a narrow sinuous fundus, and an abundant secretion. The scars in the two affections are not dissimilar; but those of leprosy are insensitive, whereas those of lupus retain the normal sensibility of the part affected.

The brighter red colour of cancerous nodules of the larynx contrasts with the dirty red, whitish, or yellowish opaque tubercles of leprosy. Larnygeal cancer may be in its later stages associated with lancinating pain; and the irregular hard edges and irregular base of cancerous ulcers, with sanguinolent muco-purulent secretion, form a marked contrast with the superficial, dry, and painless leprous ulcer.

Such, according to De la Sota, are the main points of distinction between the various diseases which may simulate leprosy of the throat.

Treatment, as a rule, can only be palliative, and is chiefly necessary in the stage of ulceration, when alterative and antiseptic solutions may be useful.

De la Sota has obtained improvement by the application of a 1 per cent solution of resorcin and of iodoform dissolved in ether, and by touching the diseased areas with a 10 per cent solution of chloride of zinc. Dr. George Mackern has had favourable results with the galvano-cautery in destroying the tubercles, especially those of the face and mouth; the eschars soon healed and the tubercles were not reproduced. When laryngeal stenosis gives rise to severe dyspnoea tracheotomy should be performed.

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loose one, and, especially when the question of treatment arises, it must not be forgotten that a good many cases occupy intermediate stages of the disease. Sometimes we see tertiary lesions in the pharynx, and then, years after, very similar lesions in the larynx, long after the pharyngeal lesions have healed. The observations of Professor Lewin of Berlin have shewn that in 20,000 cases of syphilitic affections which came under that author's observation during seventeen years in the syphilis wards of the Berlin Charité Hospital, in only about 3 per cent was the larynx attacked, and that of this number again the great majority (namely, about 87 per cent) belonged to the earlier and slighter stages of the disease; whilst in a small minority only (namely, in 13 per cent) were graver lesions found.

Syphilis of the larynx manifests itself in the following forms: (i.) Simple catarrh (Lewin's erythema); (ii.) Papules (condylomas, mucous patches); (iii.) Diffuse infiltration; (iv.) Gumma; (v.) Ulceration; (vi.) Fibroid metamorphosis; (vii.) Cicatrices, membranous adhesions; (viii.) Neoplasms; (ix.) Perichondritis; (x.) Paralyses.

Of these the first two are most frequently met with in the earlier and so-called "secondary" stages. Ulcerations are common to all stages, whilst the remainder belong to the group of "tertiary" phenomena. Lastly, all these lesions may occur in the inherited forms of the disease, though in congenital laryngeal syphilis the graver manifestations are decidedly rare.

The primary lesion is practically never seen in the larynx (though one case is reported by Moure), owing to the deep and inaccessible situation of the parts.

Syphilitic catarrh may occur as soon as six or eight weeks after the initial sore, and even while the latter is still in existence. Often it is associated with general secondary lesions. On the other hand it may appear two or three years or more after the infection, and continually recur for years, with more serious manifestations of the disease elsewhere. In no way does it differ in aspect from simple non-syphilitic catarrh, though it is remarkable for its persistency. We have not observed the distinction of colour insisted on by some observers, who allege that the colour is more dusky in syphilitic than in simple catarrh. The history and simultaneous appearance of syphilitic lesions in other parts—for instance, roseola and papular eruptions on the skin, or mucous patches on the tonsils and soft palate—generally lead to a correct diagnosis; though even when these are absent, the persistency of syphilitic catarrh and its resistance to the usual treatment for simple catarrhal laryngitis will arrest attention and lead to the suspicion of syphilis.

Mucous patches and condylomas are not often seen; in fact their occurrence in the larynx has been contested, and certainly the papular syphilide is one of the rarest forms in which syphilis appears in the larynx. Circumscribed grey thickening of the infiltrated mucous membrane may occur on the epiglottis, especially on its lingual surface;

or on the arytaeno-epiglottic folds, posterior commissure, or the vocal cords. The patches are generally single, or, if multiple, are not symmetrical. Superficial erosions—yellow, oval, circumscribed, and surrounded by an areola—may follow as the softened epithelium is abraded. As in the case of other secondary forms of syphilis, the specific catarrh and the other lesions just described tend to recur again and again for years. The symptoms are hoarseness and sometimes slight expectoration.

Of the so-called tertiary forms, *diffuse infiltration* leads to tumefaction, which usually attacks the epiglottis, vocal cords, or inter-arytaenoid fold, and sometimes causes considerable distortion of the affected parts, resulting in hoarseness and sometimes even in dyspnoea. The infiltration is due to a small-celled proliferation, which on the one hand may break down when small superficial ulcers are formed, or on the other hand may become organised into connective tissue so as to lead to a fibroid metamorphosis.

Gummas, before breaking down, are sometimes seen as smooth, red, or yellowish defined swellings, generally single, and occupying the epiglottis—especially its margin or the laryngeal surface—the arytaeno-epiglottic folds, the posterior wall of the larynx, or the ventricular bands; or they may be infraglottic. Histologically they are very similar to the diffuse infiltrations, but represent a more sharply circumscribed round-celled proliferation, developing as a rule in the submucous tissue, and thence extending towards the surface; so that the cartilages are only affected in the later stages, if at all. Very rarely does the infiltration begin in the perichondrium; if so, perichondritis may occur while the mucous membrane is still intact.

A gumma when about to break down generally becomes yellowish about the centre, ulceration follows, and the whole gumma then rapidly disintegrates from the centre towards the periphery, and a characteristic tertiary syphilitic ulcer results. (See Fig. 1, Plate XI.)

Ulcerations, if occurring in the secondary stages, are generally superficial, and most frequently are due to the breaking down of very limited and superficial inflamed areas.

Deeper ulceration belongs especially to the later manifestations of syphilis, and the ulcers present an undermined, slightly elevated, regular, sharply-cut margin, surrounded by a well-defined areola, and a floor covered by yellowish ropy mucopus and necrotic tissue. The ulcer advances more in depth than in superficial extent with resulting cicatricial contraction, and often marked laryngeal stenosis and deformity of the parts affected. (Fig. 2, Plate XI.) This is due to the well-known fact that the central portion of a syphilitic ulcer possesses the least healing capacity, and the peripheral portion the most; consequently the resulting tough scars are often more or less stellate. When this scarring occurs at the level of the glottis, or in the trachea, the resulting contraction may produce considerable stenosis and dyspnoea. Sometimes a cicatricial membranous web is formed between the vocal cords or ventricular bands, which occludes the lumen of the larynx more or less.

Inflammatory hyperplasia and oedema often arise in the neighbourhood of acute and chronic ulcers of both periods, and around the gummas, leading to exacerbations of dyspnoea and other symptoms.

Fibroid metamorphosis of the diffuse infiltration occurs in some cases, the deposit becoming transformed into connective tissue. This form, in which sometimes frequent relapses take place, each of them followed by a fibroid metamorphosis of the fresh infiltration, leads to the justly dreaded conditions of general chronic stenosis of the larynx.

Papillary excrescences or neoplasms may be found in any part of the larynx, but most frequently project from the posterior commissure. They resemble those seen in tuberculous laryngeal disease, but consist of proliferated epithelium, and closely resemble the true neoplasms. Careful examination should prevent these mammillated outgrowths from being confounded with the steep and ragged margin of a syphilitic ulcer seen in profile. A very unusual example of periodically recurring papillary growths of syphilitic origin has recently been described by one of us (F. S.). Nodular excrescences on the vocal cords appeared after inflammatory thickening and ulceration of the epiglottis and thickening of the crico-arytaenoid joints had to some extent disappeared under treatment. These thickenings subsequently disappeared; their place, however, was taken by a periodical sprouting of irregular pale excrescences, occasionally condylomatous, but more frequently papillomatous in shape, which arose sometimes more slowly, sometimes with almost incredible rapidity, from almost any part of the interior of the larynx, but most frequently from the vocal cords themselves, from the inter-arytaenoid fold, and from the ventricular bands.

Perichondritis is undoubtedly the most serious form of syphilitic disease of the larynx. It occurs in association with gumma, either by deep extension of the infiltration and, in later stages, of the ulceration, or, more rarely, by primary infiltration between the perichondrium and the cartilage, whence it proceeds upwards and downwards. In both forms necrosis and destruction or exfoliation of the cartilage attacked is apt to follow. The epiglottis is often partly or wholly destroyed in this way, whilst the arytaenoid cartilages may be expectorated, or the cricoid or thyroid cartilages laid bare till a necrosed portion comes away. But in rare cases the perichondrial infiltration, like the diffuse submucous infiltration, may escape the necrotic process, and undergo instead a form of adhesive or sclerosing metamorphosis characterised by thickening of the affected parts.

Paralysis of the vocal cords may be apparent only, and due to fibroid thickening of the perichondrium of the arytaenoid cartilages, to ankylosis of the crico-arytaenoid articulations, or to fixation of the cords from contraction in their neighbourhood. (Fig. 7, Plate VII.)

True paralysis may be of local or distant origin. The local causes are gumma in the crico-arytaenoidei postici muscles, implication of nerve fibres in syphilitic deposits, or a syphilitic neuritis; these processes may not be associated with any obvious pathological condition of the larynx.

Most frequently unilateral, these local causes of paralysis may be bilateral. Paralysis of the vocal cords may also be due to bulbar nuclear disease of syphilitic origin, or to implication of the nerve fibres in syphilitic pachymeningitis or gumma either at the base of the brain, or anywhere in their course to the larynx. In all these palsies the law of the proclivity of the abductors to succumb earlier than the adductors holds good.

The symptoms of laryngeal syphilis must obviously vary as the particular nature of the lesion, but the most remarkable feature common to them all is the almost entire absence of pain. This symptom, however, is not invariably absent, and in rare instances it has been so severe as to lead to an erroneous diagnosis of malignant disease. It is not safe, therefore, to rely too much on the absence of pain. A gumma on the posterior surface of the cricoid cartilage, for instance, may be attended with considerable pain on swallowing. In the earlier manifestations patients complain of little but hoarseness, yet when the graver lesions of tertiary syphilis appear, there is sometimes a certain degree of soreness, whilst ulceration of the epiglottis may lead to dysphagia. The peculiar "raucous" hoarse voice, or even complete aphonia, is met with in secondary as well as in tertiary cases; and if the lumen of the larynx be encroached upon by deposits or by cicatricial contraction, dyspnoea will be the result. Cough is very rarely troublesome. The dyspnoea is liable to severe exacerbations from intercurrent hyperplastic syphilitic laryngitis and oedema.

Diagnosis.—It will be seen from the description of the various manifestations of laryngeal syphilis that it is impossible to lay down hard-and-fast rules for the diagnosis of the lesions. The evidence that the *Treponema pallidum* is the specific organism of syphilitic infection is sufficiently strong to render its presence a valuable aid in the diagnosis of doubtful cases of syphilitic lesions, and is a diagnostic aid that should not be omitted where it is likely to be of service. The objective and subjective symptoms alike depend (*a*) upon the seat, (*b*) upon the intensity of the syphilitic manifestation. As syphilitic catarrh has nothing characteristic in its appearance, the diagnosis of its specific nature will be derived from concomitant syphilitic lesions in other parts, and from its submission to antisyphilitic remedies, after it has resisted mere anticatarrhal treatment. Yet, of course, the larynx of a syphilitic person may be affected by a simple laryngeal catarrh.

The diagnosis of tertiary lesions will depend on the laryngoscopic appearance, and on the history and concomitant lesions elsewhere, if any. It is, however, most important in regard to syphilis to trust to the evidence of the eye rather than to the history of the case. The patient very often does not know that he or she has been infected with syphilis; in many cases there is absolutely no history of anything to suggest this disease even when the patient is most desirous of affording all information on the point; in some cases, it is needless to say, the history of syphilitic infection is concealed.

The hoarse voice of children suffering from snuffles or broad condylomas about the anus will often lead to the suspicion that laryngeal symptoms are syphilitic. Most important is it to act on this suspicion in such patients when attacked with oedema supervening on hyperplastic syphilitic laryngitis, the symptoms being then very similar to those of membranous croup.

The two affections most likely to be confounded with laryngeal syphilis are (*a*) tuberculous disease, and (*b*) malignant disease. The distinctive characteristics of syphilitic and tuberculous ulcers have been considered on p. 198. In rare cases syphilis and tuberculous disease coexist in the larynx, in which cases there may be great difficulty in diagnosis. It must not be forgotten, too, that a tuberculous patient may acquire syphilis, and may suffer from syphilitic manifestations in the larynx, whilst his pulmonary symptoms may be due to tuberculosis, and vice versa. Again, when in syphilitic disease the lungs as well as the larynx are invaded, the real nature of the affection may be overlooked; but, first, the bacilli of tuberculosis will not be found in the expectoration; and, secondly, syphilis generally attacks the middle regions of the lungs, and, as a rule, not the apices.

In all doubtful cases iodide of potassium should be administered in considerable doses as a test of the nature of the affection. We have already alluded to the diagnostic aid afforded by the presence of the *Treponema pallidum* in doubtful cases.

As regards carcinoma, the appearances may be very similar in both affections, especially if the malignant new growth shew itself in an infiltrating form. Here again the use of iodide of potassium, a test which should be applied in all cases of a doubtful nature, will generally clear up all difficulty quickly; though it is true that a temporary subjective improvement under the use of iodide of potassium is often experienced even in malignant disease; the growth, however, steadily persists or increases in spite of the drug. Absence of glandular infiltration in the neck by no means excludes malignant disease, though, if present in considerable degree, it is more suggestive of malignant disease than of syphilis. In some cases, however, the diagnosis must remain for some time in abeyance, until careful watchfulness discovers further indications of its true nature.

Lupus may easily be confused with some syphilitic lesions; and in the absence of cutaneous lupus the difficulty in excluding syphilis, acquired or hereditary, is considerable; here again we may have to wait for the result of antisyphilitic treatment.

The significance of scars, thickenings, distortions, and webs, left after the healing of syphilitic ulcers, will generally be interpreted correctly; but sometimes, in the absence of concomitant syphilitic phenomena or characteristic syphilitic paralysis of the ocular muscles and the like, there is the greatest difficulty in deciding whether the immobility of a vocal cord is due to ankylosis from previous syphilitic disease, or to true paralysis.

Treatment.—The general treatment of syphilis of the larynx is practically the same as for syphilitic disease in other regions, and is of fundamental importance. It is necessary, however, as we have already said, to dismiss the more rigid conceptions of the so-called secondary and tertiary forms of the disease; for some cases of what would certainly be called secondary affections will only yield to iodide of potassium, whilst in tertiary lesions, on the other hand, no improvement may follow the usual course of iodides, and a cure is only to be procured by a mercurial course. Again, in other cases of tertiary syphilis, iodide of potassium produces a temporary amelioration only, and to prevent recurrence of symptoms the drug has to be continued for years. Finally, there are cases in which the alternating use of mercury and iodide of potassium produces the best results. Each case must, therefore, have its individual treatment; though no doubt the ordinary case of secondary disease is most benefited by mercury, and of tertiary disease by iodide of potassium in large doses.

In administering mercury our object should be to get the patient as quickly under its influence as possible, rather than to administer small doses over a long space of time, as advocated by some of the greatest authorities on syphilis.

The mercurial treatment recommended by Zeissl of Vienna is generally very satisfactory. Twenty grains of mercurial ointment are rubbed daily into various parts of the body: on the first day the ointment is to be applied to the skin of the neck over the larynx; on the second day, to the inner surfaces of both upper arms; on the third day, to the inner surfaces of both thighs; on the fourth day, to the inner surfaces of both forearms; on the fifth day, to the inner surfaces of both calves; on the sixth day, to the skin over both loins; and on the seventh day, to the skin of the back. This series of applications is to be repeated four or five times according to the exigencies of the individual case; each series being preceded and followed by a hot bath. In order to avoid mercurial stomatitis, astringent and antiseptic gargles and vigilant cleansing of the teeth must be used during the whole time. Zeissl's method may be adopted in all stages of syphilis.

In tertiary syphilis we give iodide of potassium, beginning with at least ten grains three times daily, and increasing this to doses of thirty or forty grains. The depressing influence of iodide of potassium is rarely observed in syphilitic cases; and that in doubtful cases the patient thrives on the larger doses is a valuable diagnostic sign, apart from any improvement in the local mischief. In other cases a combination of mercury and iodide of potassium is most suitable. If the patient belong to the wealthier classes, Aix-la-Chapelle may be recommended, because with the simultaneous use of hot sulphur baths the mercury is pushed through the system much quicker than in ordinary circumstances, and general mercurialisation is avoided. The treatment of the congenital cases is the same as that for the acquired forms, but the doses are smaller in correspondence with the ages of the patients. In obstinate cases the too long persistence in the routine administration of mercury and the iodides, especially when the disease is not controlled thereby, is not to be commended, better results will sometimes ensue on the administration of sarsaparilla in the form of Zittmann's and Kobert's decoctions. Iodipin injections also will be found useful sometimes in refractory cases.

We very rarely use any local applications to the larynx in cases of syphilis. To this general rule an exception is made in cases of obstinate catarrh; and the foul ulcers of tertiary syphilis may require some mild antiseptic spray, whilst insufflations are sometimes useful in necrosing perichondritis. But in the great majority of cases constitutional treatment only is required; under it the local manifestations will heal quickly without local measures.

Stenosis of the larynx may be due to acute lesions, such as gumma with oedematous inflammation; and tracheotomy may be demanded. Yet as a rule energetic antisyphilitic treatment will soon obviate the necessity for relief in this way.

In chronic stenosis of the larynx, such as is due to chronic hyperplastic thickening, the formation of membranous webs, and so forth, tracheotomy or intubation may ultimately become unavoidable. As a rule, tracheotomy is to be preferred, as syphilitic stenosis is liable to recur after dilatation by Schroetter's bougies or by intubation tubes. O'Dwyer, however, has most successfully treated some very obstinate cases of extreme syphilitic stenosis by dilatation. Often only small tubes can be passed at first, but after leaving these in for twelve or twenty-four hours he finds it is generally possible to introduce larger ones, and eventually to obtain a permanent stretching of the cicatrisation.

Cicatricial web formations should be divided by cutting dilators, and intubation tubes worn till the edges have healed, so as to obviate reunion and reformation of the web. But the stenosis is very likely to return after a shorter or longer interval. In a few cases thyrotomy or even laryngotomy and excision of the whole scar has been successfully performed in such circumstances, in order to enable patients to dispense with the cannula.

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Perichondritis.—*Causes.*—Perichondritis may be primary or secondary, but a primary origin is extremely rare. No doubt the vast majority of cases are secondary, although the immediate cause may be very obscure, and not infrequently, indeed, can be determined on post-mortem examination only.

Of the causes of secondary perichondritis the chief are syphilitic, tuberculous, and malignant disease of the larynx; septic inflammation; typhoid and typhus fever; variola, diphtheria, and other acute infectious fevers; gout; injuries, including wounds and blows on the larynx; scalds; and those cases in which perichondritis is set up by the impaction of foreign bodies in the larynx, by swallowing hard masses of food, by the pressure of the larynx against the bodies of the cervical vertebrae in the continual dorsal decubitus of old people, or by the frequent passage of oesophageal bougies.

Pathology.—As a result of the scanty vascular supply of the perichondrium, and the absence of vessels in the cartilage itself, the separation of the perichondrium from the underlying cartilage by inflammatory exudation often results in suppuration followed by rupture of the abscess externally with exposure and necrosis of the whole or part of the cartilage—*suppurative perichondritis*; and undoubtedly this is the usual consequence of acute laryngeal perichondritis. Yet these very peculiarities in the vascular arrangement of the perichondrium and its cartilage would lead us to expect that the less acute forms of perichondritis should be followed by inflammatory degeneration rather than by inflammatory secretion, suppuration, and consequent necrosis. Thus we have a ready explanation of the relatively uncommon, but yet by no means rare *adhesive perichondritis*, to which attention was drawn by one of us (F. S.) in 1880; in this form, without any free exudation between the inner layer of the perichondrium and the cartilage, the affected part becomes considerably thickened in consequence of an inflammatory new formation of dense connective tissue.

If an abscess form, it may rupture into the larynx, pharynx, trachea, or oesophagus; or it may discharge externally and form a laryngeal fistula. The exposed cartilage may maintain a chronic inflammation and discharge of pus for years; but sooner or later the necrosed cartilage is usually exfoliated, and may be coughed up or swallowed. When it has separated, the parts fall together and great deformity of the larynx may be brought about. Laryngeal affections in typhoid fever generally take the form of ulceration in the inter-arytaenoid fold; probably some forms of perichondritis are secondary to these ulcerative processes, whilst in others it may be a primary process; but in both cases the disease usually attacks the posterior surface of the cricoid cartilage. In tuberculosis the extension is most commonly from the arytaenoid cartilages. If the arytaenoidal perichondrium is involved, whether the inflammation take the suppurative form with consequent necrosis of the cartilage, or the more chronic adhesive form, the result is nearly always thickening of the capsule by dense connective tissue with or without degeneration or destruction of the crico-arytaenoid joint, and with more or less complete mechanical fixation of the corresponding vocal cord. The strengthening of the capsular ligament of the crico-arytaenoid joint externally by the tissue of the neighbouring perichondrium of the cricoid cartilage explains the very frequent occurrence of this mechanical impairment of the movements of the joint, even when the arytaenoid cartilage itself does not seem to have participated in the obvious inflammation of the cricoid cartilage. In course of time a relatively slight degree of thickening of the capsule, which has nevertheless been attended by fixation of the arytaenoid joint, may by contraction become less and less obvious; so that in such cases it is almost impossible from the laryngoscopic appearance to say whether a lasting immobility of a vocal cord is nervous or mechanical in origin. One of us (W. W.) has observed such a condition following diphtheria in a case in which the mechanical fixation had been erroneously attributed to pressure on the left recurrent nerve by an aneurysm.

Symptoms.—Acute perichondritis may be ushered in with a sense of chilliness, or in some cases by a severe rigor, followed by a rise of temperature and other symptoms of febrile disturbance. In other cases the onset is less acute, and the earliest manifestation may be no more than a dull aching in the laryngeal region increased by pressure. If, as is usually the case, the posterior surface of the cricoid cartilage is involved, painful deglutition is often a prominent symptom.

Dyspnoea results from excessive tumefaction on the internal surface of the thyroid or cricoid cartilages, even if it be only unilateral or from bilateral fixation of both vocal cords in or near the phonatory position; in which case the voice may be preserved while the dyspnoea is urgent. Hoarseness is by no means necessarily present; but the diseases leading to perichondritis will as a rule affect the vocal cords also in greater or less degree, and thus hoarseness or even aphonia will be present in the majority of cases. In the secondary forms of perichondritis, which as we have said constitute the vast majority, the symptoms are generally more or less modified by the primary affection; especially is this the case in tuberculosis and malignant disease, and in the acute infectious fevers with mental dulness and general apathy. Syphilitic perichondritis, like all syphilitic affections of the larynx, is seldom painful. Objectively in the early stage the only alteration in the laryngoscopic appearance may be a smooth, or irregular, nodular, unilateral inflammatory swelling, with or without immobility of the corresponding vocal cord according to the part implicated (Fig. 6, Plate VII.). If the inner surface of the thyroid cartilage is the seat of the inflammation, the ventricular band may be pushed up, forming a smooth tumefaction. If the cricoid cartilage is involved, there will be a subglottic swelling or tumefaction in the inter-arytaenoid fold, or on its posterior surface, according to the part implicated.

When the arytaenoid cartilage and its capsule are affected, they are generally red and swollen, but the tumefaction is not always obvious. Luxation of the crico-arytaenoid joint is sometimes observed. Permanent ankylosis of the joint, or at least mechanical fixation, is the usual consequence (see p. 221, and Fig. 7, Plate VII.). Frequently acute oedema of the neighbouring mucous membrane is superadded to the process taking place in the deeper tissues (Fig. 6, Plate VII.).

If the exudation and swelling do not undergo resolution, suppuration with necrosis of the cartilage may occur, and crepitus may be felt in manipulating the larynx; when the abscess has discharged, the bare necrosed cartilage may be detected with the aid of a laryngeal probe. In such cases purulent exudation, often associated with formation of fistulous tracts opening outside or into the oesophagus or other organ, may persist for years until the necrosed sequestrum is exfoliated; during this time the patient often presents a miserable aspect, and becomes greatly emaciated from the pain and dysphagia and want of sleep, and in syphilitic cases especially the accompanying fetor is often very pronounced. Sometimes large pieces of dead cartilage are coughed up; in a few recorded instances such pieces became impacted in the larynx itself, and, acting like other foreign bodies, caused dangerous dyspnoea. This may happen especially in enteric fever. Often the disease ends fatally, particularly in cases of perichondritis from malignant or tuberculous mischief; should recovery occur in other forms of perichondritis, cicatricial contraction and marked deformity and stenosis of the larynx are the too common consequences, and the bilateral fixation of the vocal cords in the phonatory position may entail dangerous dyspnoea.

The diagnosis of perichondritis of the laryngeal cartilages presents many difficulties; for obviously in the earlier stages it will often be impossible to say definitely that the inflammatory exudation and swelling involve tissues deeper than the mucous membrane; and this difficulty does not always disappear when suppuration has occurred, unless crepitus can be felt or the bare cartilage detected. In tertiary syphilitic disease especially, we often have to wait the advent of definite signs of necrosis to determine the implication of the perichondrium; in tuberculosis, suppuration, apart from perichondritis, is rare.

The painful tumefaction with deep ulceration, and possibly profuse haemorrhage, with general emaciation and constitutional weakness of advanced necrosis of the laryngeal cartilages, may be mistaken for malignant disease; on the other hand, the difficulty of eliminating perichondritis as a mere complication of malignant disease is sometimes equally great. The clinical history, the usual limitation of perichondritis to one cartilage, and, when the abscess has discharged, the less angry aspect of the swelling, will assist us to arrive at a differential diagnosis; if serious doubts as to the real nature of the case are entertained, antisyphilitic remedies should always be administered, and a portion of the swelling removed for histological examination.

The *prognosis* of laryngeal perichondritis is distinctly unfavourable, both as regards the restoration of the voice and the patency of the respiratory channel; moreover, the dangers to life are not inconsiderable. In the milder forms of adhesive perichondritis the movements of the vocal cords are rarely left unimpaired, whilst fixation of one or both cords results in marked alteration in the character and strength of the voice, and often in considerable dyspnoea. In the graver suppurative variety the patient may succumb to the disease before exfoliation of the sequestrum has occurred; and, even if he survive, the subsequent stenosis of the larynx very frequently necessitates tracheotomy and the permanent retention of the cannula.

Treatment.—At the outset, during the stage of acute inflammation, cold should be applied externally to the region of the larynx, either by the ice-bag or by Leiter's tubes; and ice should be sucked. Leeches may sometimes be used with advantage on the affected side. The patient should be kept absolutely at rest, in the recumbent position if the thyroid or arytaenoid cartilages are affected; if, however, the posterior surface of the cricoid cartilage be the seat of inflammation, the patient should lie on his side; in no circumstances should he be allowed to speak or whisper, so as to ensure absolute functional rest of the parts as far as possible. If the temperature be raised, three or four grains of quinine may be given at intervals; and for pain, if considerable, opium is useful. Food should be cold, soft, and bland. The bowels should be freely moved. If the cricoid is affected and odynphagia is very pronounced, it is better to feed the patient by rectal enemas for a few days when it may still be hoped that active antiphlogistic measures may avert suppuration. In syphilitic cases iodide of potassium should be given internally, in considerable and increasing doses; and mercurial inunctions should be made in the laryngeal region externally.

There is always a danger of acute oedema supervening, with rapid increase in dyspnoea; when other measures have failed, asphyxia may sometimes be averted by intubation or by cocaine and adrenalin applications, followed if necessary by scarification of the oedematous tissues; but if these means fail, tracheotomy must be performed.

When suppuration has occurred, with consequent necrosis, the dangers are considerably increased; the patient's strength must be maintained by tonics and generous diet. As soon as possible the abscess should be evacuated.

After the acute stage has passed, the necrosed sequestrum should, if possible, be removed; for it tends to maintain conditions which are extremely adverse to the patient's health, and, as already stated, may become dislodged and either impacted in the glottic opening or pass into the trachea and bronchi with resulting septic pneumonia. Other procedures, such as intubation and dilatation of laryngeal stricture, thyrotomy, removal of thickened parts, and so on, even laryngectomy, will also come under consideration. (See "Stenosis of the Larynx," p. 224.) If the perichondritis be due to impaction of a foreign body in the larynx, it may become necessary, even during the acute stage, to perform thyrotomy and remove the offending substance. In a case observed by one of us (F. S.) this was done with complete success by the late Sir William MacCormac. In cases of fibroid stricture thyrotomy with extensive removal of the obstructing tissue has recently yielded very satisfactory results in several cases.

Diseases of the Crico-arytaenoid Joint.—*Inflammation, Ankylosis, and Luxation.*—When we call to mind the physiological functions of the crico-arytaenoid joint, namely, those of respiration and phonation, we may almost describe it, despite its small size, as one of the most important joints in the body.

Attention has already been directed to the very frequent implication of the capsule of the joint and its articular surfaces in perichondritis affecting the cricoid and arytaenoid cartilages, which results either in suppuration and destruction of the joint, or in adhesive inflammatory degeneration with thickening of the capsule or true ankylosis of the joint.

Definition.—We call every degree of stiffness of the crico-arytaenoid joint, which is produced by mechanical causes, an ankylosis of this joint; and we distinguish two forms, namely, first, the *true ankylosis*, in which the stiffness is produced by intracapsular disease; and, secondly, the spurious or *false ankylosis*, in which extracapsular changes lead mechanically to impairment of its functions. In some cases true ankylosis is a consequence of a long-existing false one.

Luxation of the crico-arytaenoid articulation, first described by B. Fränkel, means a displacement of the arytaenoid cartilage from the articular surface of the cricoid; in some cases both ankylosis and luxation coexist.

Causes.—Every true ankylosis is the product of an inflammatory degeneration of this joint, however slow and insidious the degenerative process may have been. The possible causes of ankylosis of the joint are as follows:—

(*a*) Ankylosis from local inflammatory causes, namely: Perichondritis, suppurative or adhesive (by far the most frequent cause); simple plastic laryngitis (?); lesion of the joint by wounds, ulceration, luxations, contusions, and congenital causes.

(*b*) Ankylosis from constitutional causes leading to local affections, namely: enteric fever, variola, syphilis, diphtheria, tuberculosis, gout, and excess of the physiological senile ossification.

(*c*) Ankylosis from purely mechanical causes leading to permanent immobility, namely: Cicatricial contractions of the mucous membrane or of the muscles after injuries, enteric, syphilitic and other ulcerations (false ankylosis), neuropathic or myopathic paralysis, diaphragms or complete subglottic obliteration of the laryngeal passage, neoplasms.

The symptoms will depend, first, on the position taken by the arytaenoid cartilages, and consequently by the vocal cords; and, secondly, on the amount of tumefaction and inflammation in and around the crico-arytaenoid joint. Thus the joint may be fixed in any position, from that of deepest inspiration to that of phonation; and the arytaenoid cartilage may be drawn even across the median line. These extreme positions are mostly found as the result of cicatricial contraction after syphilis and other ulcerative diseases; whilst in true ankylosis the implicated cord generally varies in position from the phonatory to what is called the cadaveric position, which lies midway between phonation and deep inspiration.

If bilateral ankylosis have occurred, the fixation of the cords is not necessarily symmetrical.

Tumefaction is obvious in the majority of cases of true ankylosis; but in the spurious cases, even if true ankylosis should eventually supervene, this sign may be wholly absent. In short, immobility with tumefaction favours the diagnosis of mechanical impairment; immobility without tumefaction does not exclude this possibility. The swelling is sometimes very considerable, and may in itself be a serious impediment to respiration. In complete ankylosis there will be complete immobility of the arytaenoid cartilage and corresponding vocal cord; in incomplete ankylosis the mobility will be either restricted or jerky.

When ankylosis is combined with luxation of the joint, the position of the arytaenoid cartilage will be abnormal, in addition to the swelling and immobility. In simple luxation the appearances are very similar to those presented by the last-mentioned combination, except that it is possible to reduce the luxation.

The chief subjective symptoms are alterations in the voice and dyspnoea. Each is determined by the position in which the affected vocal cord or cords are fixed. The quality of the voice may be unaltered, whilst in all other cases it may be completely lost; more usually hoarseness, weakness, or diplophonia are observed. Dyspnoea occurs when both vocal cords are fixed near one another.

These symptoms are met with in infinite variety and degree; but they are so frequently modified by the primary disease that we can only draw attention to the main features.

Diagnosis.—When we consider the great variety of symptoms and objective appearances due to ankylosis, or associated with it, which may be encountered, and the numerous complications that so frequently coexist, it is easy to understand that in many cases a definite diagnosis of the condition cannot be made. More especially is this true of those cases which, not being associated with any obvious thickening of the arytaenoid joint, exactly simulate palsy of the vocal cords of nervous or myopathic origin; on the other hand, the tumefaction of ankylosis may be mistaken for extensive effusion into and swelling of the soft parts covering the cartilaginous framework, with resulting temporary spurious ankylosis.

The most important points in favour of the diagnosis of ankylosis are the presence of tumefaction around an immobile arytaenoid; abnormal position of the arytaenoid cartilage; unilateral distortion of the contour of the larynx from cicatricial contraction or luxation; fixation of the vocal cord in the abducted position.

The prognosis, as to life, will depend on the nature of the primary disease, on the amount of tumefaction, and on the position assumed by the vocal cords; for instance, a position of bilateral abductor paralysis with the cords in the phonatory position is liable to end abruptly by acute asphyxia.

As to recovery of function, we must regard the length of time the ankylosis has existed, and again the nature of the primary disease. False ankylosis is more hopeful than true ankylosis; but if either have existed for a few months, very little hope can be entertained of complete recovery.

Treatment.—If the patient's life be in danger from asphyxia it may be necessary to perform tracheotomy before any measures for the treatment of the ankylosis can be undertaken; indeed, the nature of the primary disease may be such as to claim our entire attention, or may preclude the possibility of any successful therapeutic measures directed to the crico-arytaenoid joint.

On the other hand, the subjective symptoms may be unimportant and unattended with any inconvenience; in this case it is better to leave well alone rather than run any risk of importing fresh and perhaps dangerous complications. Especially is this the case when true ankylosis has existed for a considerable time; then indeed treatment is useless.

Thus the indications for operative treatment are limited to the cases in which dyspnoea is a prominent symptom, in which there is no evidence of true ankylosis of the joint of long standing. We may then hope to obtain relief by mechanical dilatation by means of von Schroetter's bougies, or by O'Dwyer's method of intubation continued for a long time.

But more help can be afforded by early methodical dilatation in preventing the occurrence of extreme stenosis and cicatricial contraction or luxation; and by timely treatment of more recent cases due to typhoid fever, syphilis, or to perichondritis from other causes.

Cheval states that he was able to reduce a recent simple luxation of the joint by means of a strong faradic current, a double electrode being applied to the posterior wall of the larynx so as to tetanise the inter-arytaenoid and posterior crico-arytaenoid muscles.

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Stenosis of the Larynx.—*Causes.*—Laryngeal stenosis may be due to various causes, namely:—

i. Infiltration of the tissues of the lining membrane, (*a*) by inflammatory exudation or oedema in the course of acute catarrhal or septic inflammations, scalds, typhus or typhoid fevers, measles and other exanthems, syphilis, tuberculosis, perichondritis, wounds, scalds, and other injuries; or (*b*) by gummatous deposit, tuberculosis, cancer, lupus, or leprosy.

ii. False membranes in croup and diphtheria.

iii. New growths, either benign or malignant.

iv. Congenital webs or adhesions between the vocal cords, cicatricial contraction following syphilis, lupus, perichondritis, typhoid fever, wounds, and so forth.

v. Bilateral abductor paralysis of the vocal cords, whether neuropathic or myopathic in origin, or mechanical fixation of the cords in the phonatory position.

vi. Foreign bodies.

The occurrence of stenosis of the larynx has been incidentally referred to under the above-mentioned diseases, so that its characteristic symptoms and laryngoscopic signs need not be related again. Here we shall confine ourselves to the intra-laryngeal operations for the relief of laryngeal stenosis, namely, intubation, dilatation by bougies, and so on, without reference to the question of the removal of the obstruction in the case of foreign bodies or new growths, or to the various antiphlogistic procedures and scarification that have been fully discussed elsewhere.

Acute Laryngeal Stenosis.—In acute stenosis requiring operative interference the choice lies between tracheotomy and intubation. If stenosis of the trachea be present at the same time, as in the case of diphtheritic membranes which have extended down the trachea and bronchi, or of compression of the trachea by an aneurysm or growth which has also caused bilateral abductor paralysis of the larynx, the question whether the dyspnoea will be relieved by any operative procedure confined to the larynx will arise. For the method of performing tracheotomy the reader should consult text-books of surgery.

Intubation of the Larynx.—It is to Leopold von Schroetter of Vienna and to Joseph O'Dwyer of New York that we owe the instruments whereby this method of treating laryngeal stenosis has been made practicable. Schroetter's method of effecting dilatation by the introduction of hollow vulcanite tubes, or of solid zinc-bougies (when tracheotomy has been practised previously), has at present been practically superseded by that of O'Dwyer. The latter's tubes for children are made of gilt metal, varying in length from 1½ inch up to 2½ inches for the age of twelve; the longer and larger tubes for adults are made of vulcanite. When the tube is in the larynx a flange at the upper end of it rests on the ventricular bands, and the rest of the tube lies below the vocal cords.

In proceeding to intubate young children the patient is closely wrapped in a blanket with the arms included, and is held sitting upright on the nurse's lap facing the operator. The mouth is kept open, and the head held steadily in the vertical position by an assistant. The operator, having passed the left forefinger into the larynx, hooks forward the epiglottis; the tube suited to the patient's age is then rapidly introduced on the obturator, which is attached to a handle held in the right hand, and is guided into the larynx by the finger which is hooking up the epiglottis; then the sliding rod on the handle is made to disengage the tube from the obturator, which is at once withdrawn, the left forefinger meanwhile fixing the tube and retaining it in position; finally this forefinger is removed. In the hands of a skilful operator the whole procedure occupies from three to five seconds. The tube should now be in position; but should it have been inadvertently passed into the oesophagus it may be extracted at once with the silk-thread loop attached previous to introduction. When the tube has been properly introduced the child gives a few strong coughs at first; but in the course of a few seconds the breathing is manifestly relieved, and the larynx very soon tolerates the tube. The loop should not be removed for ten minutes, as it tends to induce the coughing up of mucus and sometimes of small pieces of false membrane. Unless false membrane or other causes of obstructive dyspnoea exist in the trachea or bronchi, the embarrassed respiration gives place to quiet breathing, and the patient, who should always be placed in the steam bed, drops off into a calm sleep. Of course the patient is completely aphonic so long as the tube remains in the larynx; and though it removes glottic obstruction to breathing at once, inasmuch as the glottis cannot be closed, it acts exactly like a tracheotomy tube in rendering coughing less effectual.

The tube may be left undisturbed for five days, or more if necessary, but sometimes it becomes more or less blocked by false membrane; in other cases it may be desirable, though rarely necessary, to remove the tube occasionally to allow the patient to clear the lower air-passages of tenacious mucus. For this purpose, and in order to enable the patient to imbibe a large amount of liquid food without discomfort, one of us (W. W.) makes a practice of removing the tube daily in older and docile children; for in them both intubation and extubation are rapidly accomplished without resistance, or the slightest risk of injury to the larynx.

Extubation is more difficult than insertion of the tube. For this purpose the child is placed in the same position as for introduction, and expanding forceps, specially made for the purpose, are guided into the upper orifice of the tube by the left forefinger, previously passed into the larynx, till the instrument impinges on and fixes the posterior border of the flange; they are then opened so as to hold the tube firmly while it is rapidly withdrawn. Neither intubation nor extubation should occupy more than fifteen seconds at the outside, as respiration is necessarily suspended during each process: if therefore an attempt be not promptly successful, it is better to try again rather than, by prolonged manipulation, to run any risk of asphyxia, or of setting up exhausting struggles for breath. It need hardly be said that no force should ever be used.

If false membranes be present in the larynx the thread should not be cut short, but looped over the ear and fixed by plaister; or O'Dwyer's short tubes specially made for these cases should be used. These are short hollow cylinders of large calibre, which do not push the false membrane down. As they have no retention-swell it is necessary to use the largest size possible. The symptoms of false membrane are sudden obstruction to the out-going air in expiration, and especially a flapping sound in coughing and a croupy cough when the tube is in.

The greatest care in feeding the patient is necessary to prevent the escape of food into the trachea. By intelligent children soft, semi-solid food can often be gulped slowly without risk. Liquid food may be taken if the patient can be induced to suck it through a tube, or to take it from an ordinary feeding-bottle while lying face downwards on the nurse's lap, or on a bed with its head pendent. If this does not answer, the patient must either be fed through a nasal tube, or nutrient enemas must be given.

Intubation should be performed early so as to prevent the engorgement of the lungs and the pulmonary collapse consequent on prolonged dyspnoea.

The advantages of intubation over tracheotomy in the treatment of acute laryngeal stenosis are:—

(*a*) Its simplicity and painlessness, well exemplified by a case under one of us (W. W.), a child seven years old, who, having on former occasions experienced intubation and extubation at his hands, sat up and enabled him to extract the tube without being held or in any way restrained. On account of the relatively simple character of intubation, we can resort to this procedure much earlier than tracheotomy, and thus avoid all "cutting," to which parents sometimes will not consent.

(β) In children under five years of age the percentage of recoveries is considerably higher than after tracheotomy.

(γ) The intubation tube is more comfortably worn than the tracheotomy tube, in fact when in place it cannot be felt at all.

(δ) Respiration is conducted through the natural passages.

(ε) No anaesthetic is required as a rule, though cocaine may be used with great advantage. If the patient struggle much, especially in the case of an older child, chloroform should be given, and intubation or extubation performed in the recumbent position, rather than run any risk of exhausting the patient or of injuring the larynx.

The following difficulties may arise:—(i.) False membranes may occasionally be disengaged and crushed down into the trachea on introducing the tube. In such an event the tube can be withdrawn at once by the attached loop and the loosened membrane expectorated. (ii.) The tube may be coughed out and the dyspnoea return before help can be obtained. (iii.) Asphyxia may occur from blocking of the tube by false membrane. Such an accident can only occur in very feeble patients, as the tube, if blocked, is always expelled at once by a vigorous cough. Asphyxia may also arise from oedema above the tube, but it is a very unlikely occurrence. (iv.) Ulceration at the cricoid ring or of the walls of the trachea may be caused by an ill-fitting tube. (v.) Careless and rough introduction may make a false passage. (vi.) If the extubating forceps be opened widely outside the orifice of the tube as it lies in the larynx, instead of within it, the tissues may be torn as the instrument is withdrawn. (vii.) Difficulty may arise from subglottic stenosis at the narrowest part of the respiratory passages—the cricoid ring; but if the tube will not readily pass the obstruction here, a smaller one should be used. (viii.) If special precautions are not taken, "foreign-body pneumonia" may arise from inspiration of liquid food. (ix.) Temporary aphonia sometimes persists for a few weeks after removal of the tube.

Intubation is chiefly practised for the relief of acute laryngeal stenosis in diphtheria and membranous croup; but it is sometimes to be recommended in recent cases of crico-arytaenoid fixation following typhoid fever, syphilis, and perichondritis from other causes; methodical dilatation by means of O'Dwyer's tubes or Schroetter's bougies may also be employed to prevent the occurrence of cicatricial contraction.

One of us (W. W.) has observed several cases of acute laryngeal stenosis in adults (due to inflammatory swelling from various causes) in which a tracheotomy otherwise inevitable was obviated by intubation. We should also be disposed to try it in acute septic oedema of the larynx.

It is impossible from statistics alone to draw comparisons between tracheotomy and intubation; for whereas in diphtheria and acute inflammatory affections, at any rate, intubation is or should be adopted as soon as urgent dyspnoea is found not to be relieved by the use of the steam bed, calomel fumigations, and other means, tracheotomy is always delayed as long as reasonably possible. On the other hand, the early relief of intubation undoubtedly saves many lives that would otherwise be sacrificed, not by asphyxia but by pulmonary engorgement and lobular pneumonia. Before the introduction of the diphtheria antitoxin, statistics shewed that under the age of five the results of intubation are better than those of tracheotomy; after this age the percentage of recoveries was *slightly* in favour of tracheotomy up to the twelfth year; whilst above the age of twelve tracheotomy yielded much better results.

But by the use of diphtheria antitoxin, not only has the mortality in cases of laryngeal diphtheria been very materially decreased, but with the relatively rapid relief of the laryngeal obstruction the difficulties and dangers of intubation have greatly diminished. The tube can in many cases be permanently removed in forty-eight hours, and not infrequently after a much shorter period. We should, therefore, give the preference to intubation over tracheotomy whenever it is practicable—secondary tracheotomy, speaking generally, being reserved for cases in which, for any reason, intubation has failed to give relief.

On the other hand, it should be borne in mind that the favourable influence of the diphtheria antitoxin injections on the results of intubation extends equally to tracheotomised patients, and that, inasmuch as the tracheotomy tube can often be safely discarded within a very few days, many of the secondary complications arising from tracheotomy are likewise avoided.

Chronic Laryngeal Stenosis.—In cases of chronic laryngeal stenosis, in which the cause of the obstruction cannot be removed, tracheotomy is generally preferable to intubation, inasmuch as the latter entails loss of the voice, and the patient can only speak in a whisper; whereas after tracheotomy the patient very soon gets into the way of stopping the tracheotomy tube with his finger while speaking, and may continue to wear a tube for thirty years or more without discomfort. In addition, methodical dilatation in cases of chronic stenosis is an extremely tedious process, and recurrence of the stenosis, when the dilatation has been discontinued, unfortunately is anything but exceptional.

On the other hand, v. Schroetter, O'Dwyer, and others have obtained brilliant results in several cases of stenosis, from chronic cicatricial contraction of the glottis following syphilitic disease, typhoid fever, injuries, etc. Often the tubes which can be passed at first are very small; but after leaving these in for twelve or twenty-four hours, it is generally possible to introduce a larger size, and so by patience and perseverance the largest tube can ultimately be passed; thus the cicatrix is more or less permanently stretched, and the dilatation can be maintained by passing a large tube once in three months.

The use of Schroetter's zinc bougies over a long period is sometimes successful in producing sufficient dilatation to obviate the further necessity for wearing a tracheotomy tube; in other cases, especially of membranous cicatrices between the vocal cords, intubation or mechanical dilatation, after section of the web by a cutting dilator, will yield favourable results: thyrotomy, with resection of the cicatricial tissue, is an alternative procedure.

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New Growths

Benign Growths.—**Causes.**—Although benign growths of the larynx are of fairly common occurrence—a fact well demonstrated by the collective investigation instituted by one of us (F. S.), which resulted in bringing together no less than 10,747 cases observed by 107 laryngologists between 1862 and 1888—yet it is very difficult to assign their occurrence to any particular or definite causes. We are wont to look upon chronic laryngeal catarrh as the most prolific cause of innocent laryngeal tumours; but although chronic inflammatory affections of the larynx are common enough, we are not aware of any trustworthy observation of a new growth actually making its appearance in the course of a chronic laryngitis under the eyes of the observer, except, perhaps, the little inflammatory thickenings on the borders of the first and middle thirds of the vocal cords, known as "singers' nodules," often seen in singers and actors who have over-used their vocal organs. No doubt some laryngeal catarrh may be seen in association with benign growths; but this is a consequence of the presence of the neoplasm rather than the cause: moreover, in the majority of cases catarrhal processes are not present.

PLATE IX

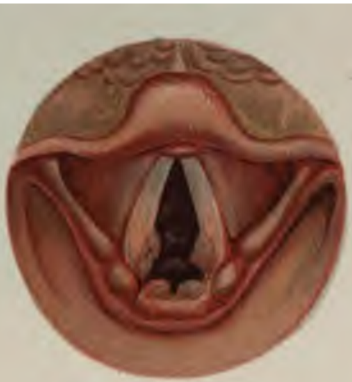

Fig. 1.

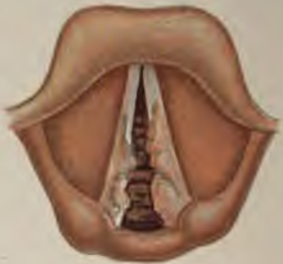

Fig. 2.

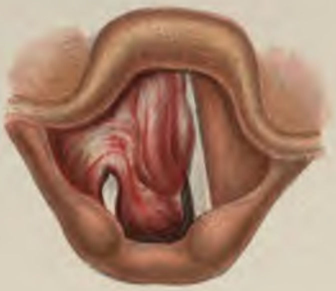

Fig. 3.

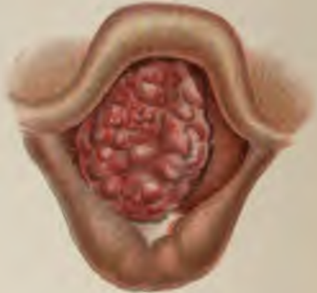

Fig. 4.

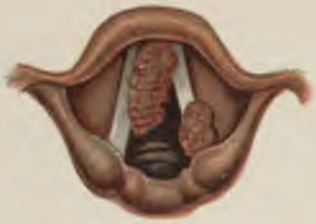

Fig. 5.

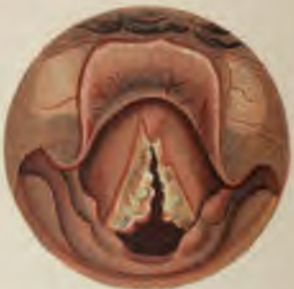

Fig. 6.

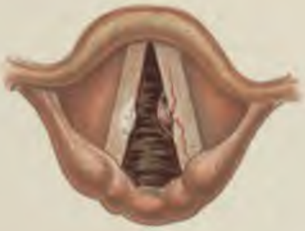

Fig. 7.

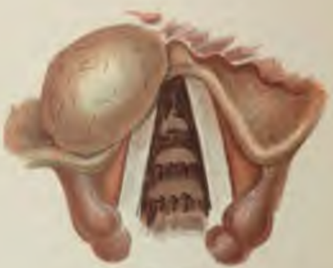

Fig. 8.

PLATE IX.

BENIGN NEOPLASMS OF THE LARYNX.

Fig. 1. Pachydermia laryngis, typical, being restricted to the vocal processes and to the inter-arytaenoid fold. (P. W. W.)

„ 2. Pachydermia laryngis, diffusa. The thickening extends over a considerable portion of the length of the vocal cords, resembling in its distribution some forms of tuberculous deposit. (P. W. W.)

Figs. 3 and 4. Large fibroma growing from the right ventricular band; during inspiration it hung down between the vocal cords (Fig. 3), and on expiration it was extruded above the glottis (Fig. 4). (P. W. W.)

Fig. 5. Papillomas laryngis. (P. W. W.)

„ 6. Diffuse papillomas of the larynx. (Krieg.)

„ 7. Fibroma of the vocal cord, subglottic. (P. W. W.)

„ 8. Cystoma of the epiglottis: no symptoms. (P. W. W.)

Again, several cases of congenital new growths in the larynx are on record, and this fact, together with the relative infrequency of laryngitis in cases of benign growths, seems to exclude the probability that chronic catarrh is an essential factor in their occurrence. Whether occupation exercises much influence in the matter is also open to discussion. Excessive use of the voice has been held responsible for the appearance of innocent neoplasms, but the very large number of benign growths occurring in young children, and their appearance not only in the newly-born but even in deaf mutes, shew that such a cause cannot be widely operative; though we frequently meet with small circumscribed thickenings of the vocal cords, chiefly in singers who over-use their vocal organs or use them improperly. Men are more frequently attacked than women; yet the difference is not so striking as in the case of malignant growths of the larynx. No time of life is free from them; but they are most commonly met with between the ages of twenty and forty; and, next to this period, the first few years of life furnish the most cases. Though there have been instances of benign growths beginning in patients over seventy years of age, it is a good rule to look with suspicion on all growths which arise after the fiftieth year, as experience teaches that growths appearing at this time of life are much more frequently malignant than benign.

As regards the various forms of benign growths in the larynx, in order of frequency they are as follows:—Papilloma, fibroma, cystoma, myxoma, adenoma, lymphoma, lipoma, angioma, ecchondroma, and growths consisting of normal thyroid tissue. Practically, of all these varieties three only are common; namely, papilloma, fibroma, and cystoma: the others are so very rare as to be pathological curiosities.

Papilloma.—This is by far the commonest variety, constituting fully 39 per cent of all laryngeal growths. It is met with at all ages, but especially in young adults. Papilloma may be single or multiple, varying in size from a millet seed to a walnut, and from a white or delicate pink to a red, granular, cauliflower-like appearance. These growths are usually pedunculated, firm in texture, and do not readily bleed (Figs. 5, 6, Plate IX.). Histologically they are composed of a number of vascular papillae, covered by an epithelial layer. Their favourite seat is on the vocal cords; and of these, again, the anterior commissure and anterior thirds are more often attacked. Next in frequency come papillomas of the ventricular bands, where they are generally observed only in cases of multiple papillomatous degeneration. Sometimes they are seen projecting from the ventricle of Morgagni; in other cases they are attached to the arytaeno-epiglottidean folds and to the epiglottis. In the latter positions they are very rare, and if observed in patients over fifty they should always be looked upon with suspicion of malignancy. Unlike epithelioma, their area is distinctly restricted; they do not infiltrate the surrounding tissue, and they are practically never seen in the inter-arytaenoid fold. Early epithelioma of the larynx may very closely resemble a benign papilloma; the differential diagnosis is fully discussed on p. 246.

In syphilis and tuberculous disease of the larynx false excrescences are frequently observed in the inter-arytaenoid fold or on the vocal processes; these and "pachydermia verrucosa" might be mistaken by the inexperienced for a benign growth if due attention were not given to their characteristic features, which are elsewhere described.

Fibroma consists of the same tissue as the vocal cords, and originates in inflammatory thickening of their fibrous basis. It consists of connective tissue with an admixture of elastic fibres, is vascular, and may contain cavernous blood-spaces. These tumours are covered by epithelium, and serous infiltrations and haemorrhages are common in them, especially in the softer varieties. The vascularity, particularly that of the sessile forms, is very considerable; and the haemorrhage on removal is often much greater than in the case of papilloma.

Fibroma occurs in two forms, sessile and pedunculated, and in both forms is generally single, with a white, pink, cherry-red, or even bluish smooth surface. It generally occupies the upper surface of the middle or anterior half of a vocal cord, and varies in size from a millet seed to a walnut. Two very extraordinary cases of soft fibroma have been reported by Paul von Bruns and Semon, in which fibromatous neoplasms of the neck entered through the cricothyroid and thyrohyoid membrane respectively, into the larynx, and in which it was possible to remove them by external operation without opening the cavity of the larynx. Semon's case was shewn before and after removal to the Laryngological Society of London, and was fully described in the *British Medical Journal* in 1905. The patient was a lady, aged forty-seven, who thirteen years previously had had tracheotomy performed on account of a huge growth filling up her whole larynx; there was a tumour the size of a walnut in the left submaxillary region, pressure on which caused retching and coughing. In 1905 an incision was made over the external tumour, which was found to be connected by a thin pedicle with the internal, and the latter was shelled out without the cavity of the larnyx being opened at all. The tracheotomy tube, which had been worn thirteen years, was removed and the tracheal wound closed. The patient made an excellent recovery and keeps perfectly well. The accompanying illustrations give a good idea of the laryngoscopic aspect and of the form and size of the tumour. The multiplicity common in papilloma is not seen in fibroma. Sessile fibroma is almost always semi-globular; in the pedunculated form the stalk may be slender or stout, long or short. Sometimes the pedicle is long enough for the growth to hang down into the subglottic cavity, and to escape from sight except on forced expiration or cough, when it may be thrown above the level of the vocal cords; whilst on deep inspiration it is sucked into the subglottic cavity, and may completely disappear in it, the vocal cords on the next phonation meeting over it, so that the slight inequality in one cord, indicating the origin of the pedicle, alone betrays its existence (Figs. 3, 4, Plate IX.). Fibromas vary greatly in size, from a pea to a hazel-nut or more.

Cystoma.—As cysts result from obstruction in the duct of a muciparous gland, they generally occur where these are plentiful, and especially on the dorsal surface of the epiglottis. But they may occur in any part of the larynx where glands exist; and then they are found on the ventricular bands, or growing from the ventricle or the arytaenoid region, and, in rare cases, even from the vocal cords. They are smooth, tense, globular, semi-translucent, covered with light red or greyish-pink mucous membrane, and, if considerable in size, have blood-vessels coursing over their surface. Sometimes they attain such a large size as to be visible with the naked eye when situated on the epiglottis; and if arising in the larynx itself they may actually threaten suffocation (Fig. 8, Plate IX.).

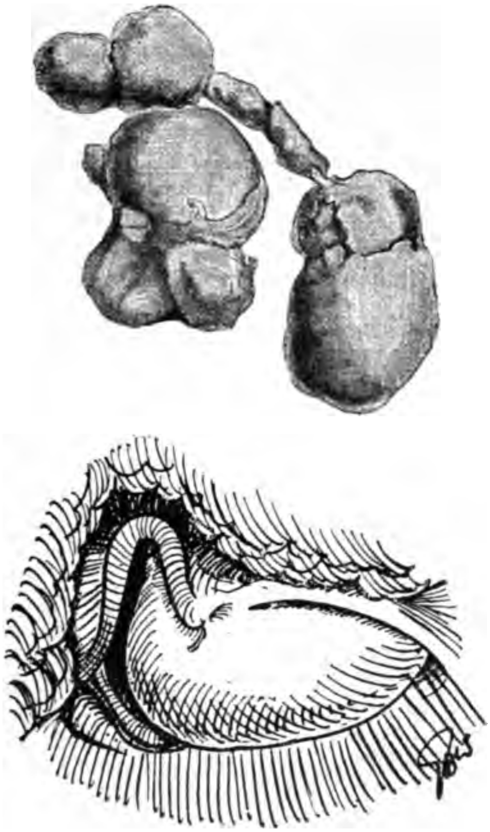

FIG. 29.—The lower illustration shews the laryngoscopic image of a fibroma which was subsequently removed without opening the larynx. The upper illustration shews the tumour reconstructed after removal.

The other forms of laryngeal benign growths, as already stated, are very rare; some of them, such as lymphoma and mycosis fungoides, need only be mentioned by name.

Angioma is generally unilateral and single, occurring in the sinus pyriformis, or on the ventricular bands, vocal cords, or epiglottis, of characteristic aspect, and composed of a mass of blood-vessels held together by a small quantity of loose connective tissue. The growth is red or purple in colour, and rarely exceeds a filbert in size. A case has been described by Semon and Shattock, in which a malignant growth, originating from the left arytaeno-epiglottidean fold, closely simulated an angioma.

Myxoma usually occurs on the vocal cords. It likewise is generally solitary, small, pink, or greyish-white, sessile, translucent, and well defined. If pedunculated, the growth partakes rather of the fibromyxomatous nature, and then may present a mammillated surface and resemble a papilloma in aspect.

Ecchondroma mostly arises from the cricoid cartilage. It has been observed growing from the epiglottis, thyroid, and arytaenoid cartilages. Ecchondromas are usually firmly attached, hard, sessile growths, presenting a smooth surface of irregular outline and covered with healthy mucous membrane.

Lipoma may attain considerable size. One removed by Sydney Jones from the right arytaeno-epiglottidean fold partly projected into the patient's mouth, so enormous was the size it had attained.

Prolapse of the ventricle of Morgagni, though strictly speaking not a new growth, clinically resembles a laryngeal neoplasm so closely that it may be conveniently mentioned here. A smooth, pink, lobulated, supraglottic mass, generally unilateral, sometimes bilateral, is seen resting on the vocal cords, and corresponding to the opening of the sacculus, which, being inverted, of course no longer exists. That a real inversion should be possible seems hardly credible; yet several cases have been reported both during life and on the post-mortem table by trustworthy observers. It is most frequently seen in phthisis pulmonalis. After all, the so-called inversion, at least in the cases under our own observation, is in reality an oedematous or infective swelling of the lax submucosa, which consequently causes a protrusion to take the place of the natural hollow. As it is useless to replace it, the projecting portion should be snared or excised. Anyhow it should be borne in mind that dislocation is exceedingly rare, and would be closely simulated by outgrowths from the ventricle; in fact, the occurrence of true inversion has been doubted again and again.

The **symptoms** of benign laryngeal growths, it is needless to say, will vary according to their size and situation. By far the most frequent symptom met with, and indeed in most cases the only one, is alteration of voice. The explanation of this is obvious since the vocal cords are the principal seat of these growths. The degree of vocal impairment will depend, of course, on the amount of interference with the free vibration of the vocal cords. Thus, a very small growth occupying the anterior commissure or the free border of the cords in their anterior third may greatly impair the voice or even produce complete aphonia; whereas growths which do not encroach on the free borders, or which are situated on the middle parts of the cords, may give rise to a much less marked vocal impairment; in some cases, indeed, no symptoms whatever occur.

When the growth is sufficiently large to encroach considerably on the glottic space, and to narrow the canal of the larynx, dyspnoea must result, and the degree of dyspnoea will of course depend on the degree of narrowing of the canal.

Cough is rarely a prominent symptom, but in very young children with papilloma it may be present and be croupy in character, as the growths are apt to excite some degree of laryngitis and glottic spasm. Pain is hardly ever felt, and only in a few cases, particularly of pedunculated growths, are strange sensations noticed, whilst spontaneous haemorrhages practically never occur. Dysphagia may be present when a large growth is attached to the upper surface of the epiglottis.

The **prognosis** as regards life and health is nearly always most favourable, but the possible developments which these growths may take if left untreated must not be forgotten. We have already mentioned that papillomas occur either in the solitary or multiple form. In the former case, after having attained a certain size, they may remain stationary for a long time; but they are more likely to become gradually larger, and this is, indeed, the rule with the multiple forms; in this case they encroach on the glottic space and threaten asphyxia, an event which has indeed occurred in several instances.

Fibroma, after having attained a certain size, not rarely becomes stationary; in other cases, however, it continues to grow slowly, and may sometimes, after many years, cause serious respiratory difficulties. In a case of a large pedunculated fibroma observed by one of us (F. S.), suffocation occurred quite suddenly, probably from impaction of the growth in the glottis. No post-mortem examination was obtained.

Spontaneous expulsion of new growths has been reported very rarely indeed; so excessively rare is it that the prospect of it ought not to be held out to any patient. One of us (F. S.) has seen involution take place in the course of years in a few cases of growing children with small nodules, apparently fibromatous, on their vocal cords; but this, too, is certainly very rare. On the whole it may be said that benign laryngeal growths, when left to themselves, though they may become stationary at a certain period, are more likely to increase gradually in size; and papillomas do so sometimes rather rapidly. The prognosis from the therapeutic point of view is nowadays almost universally good, although the tendency of papilloma to recur must always be remembered. The prognosis as to the recovery of voice is also, on the whole, very good, though in cases of sessile or very multiple growths some small vocal disturbances may remain behind after their removal. In some forms of benign growths the prognosis should be very guarded, if not as to life, yet at any rate as to duration of disease and to subsequent function of the parts, particularly in cases of papilloma in early childhood in which it may become necessary to perform prophylactic tracheotomy to prevent suffocation, or in which thyrotomy has been carried out for removal of the growth; also in cases of ecchondroma, which may attain such a size as to necessitate permanent retention of a tracheotomy tube or even total extirpation of the larynx.

We must here refer to a question which at one time was the subject of a good deal of controversy, namely, whether benign growths of the larynx ever undergo malignant transformation, and, if so, whether this tendency is increased by intra-laryngeal operative interference. This question could only be answered definitely by a critical review of a very large number of cases, and to this end the collective investigation already referred to was instituted by one of us (F. S.) with the following result:—Of 10,747 cases of innocent laryngeal growths observed by 107 laryngologists, 8216 had been operated on intra-laryngeally; of these in 33 cases malignant transformation was reported, that is to say, 1 transformation in 249 cases; but on critically analysing the individual cases of reported transformation, in 5 only was such transformation found to have been quite or almost undeniable; and even if 7 further cases in which the transformation was more or less probable be added to the number of the certain cases, the proportion of transformation would be but 1 in 685 cases. The remaining cases of reported transformation were of an exceedingly doubtful character, and in most of them it was probable that a mistake in diagnosis had been made from the very beginning. In any circumstances the occurrence of a malignant transformation of a previously benign laryngeal growth must be considered as an event of the greatest rarity; and the very number adduced affords sufficient evidence that the alarm which has been raised concerning the influence of intra-laryngeal operation upon the occurrence of such transformation is absolutely unfounded. A further proof of this conclusion is that a positively larger number of spontaneous transformations in non-operated cases were reported in the collective investigation than of transformations after removal; the percentage in the first class of cases was 1 to 211, in the second class 1 to 249. Since this investigation was published, in 1888, some very few fresh cases of apparent or real transformation have been reported, but the conclusions derived from it have remained perfectly intact. Of course we do not deny the possibility that benign laryngeal growths may sometimes undergo malignant transformation like benign growths in other parts of the body; but there is no evidence that this is aided by intra-laryngeal operations. It is much more probable that cases are diagnosed as benign which were really malignant from the outset.

Diagnosis.—It is needless to say that the diagnosis of benign laryngeal growth can only be made by means of a laryngoscopic examination, as the symptoms consist almost entirely of vocal impairment and perhaps dyspnoea, the former of which may equally well be due to chronic laryngitis and numerous other causes, whilst both symptoms may be produced by syphilitic, tuberculous, or inflammatory disease, or by paralytic disorders. The differential diagnosis between benign growths on the one hand and these several diseases on the other is not usually difficult, though it is sometimes impossible to distinguish between benign growths and tuberculous tumours; sometimes indeed this can only be definitely settled by a microscopical examination of the fragment removed. The appearances presented by the various forms of new growth have already been sufficiently noted, and the very important question of the differential diagnosis between benign and malignant tumours is fully discussed further on (p. 246 *et seq.*). In some rare cases blood-clots in the larynx may so closely simulate benign (or malignant) new growths, as to make the differential diagnosis previous to removal practically impossible (Fig. 8, Plate X.).

Treatment.—A very few cases of benign laryngeal growths are best left alone; they are chiefly cases of small sessile fibroma situated on the vocal cords, and causing very slight symptoms. In such cases removal is sometimes exceedingly difficult, and in the endeavour to remove them there is a risk of injuring healthy parts in the neighbourhood and of bringing about still greater vocal impairment. These cases, however, are very exceptional, and in the vast majority it is not only desirable, but even necessary, on account of the symptoms, to remove the neoplasm. Astringent local remedies have been advocated, and it has been stated that growths have been made to disappear by their use; but we have never seen such a happy consummation, and in our opinion not only is it mere waste of time to resort to the use of these applications, but they are apt to set up injurious irritation. Voltolini's method of running a little sponge attached to a laryngeal probe up and down the larynx, by which process soft growths are supposed to be torn from their attachments, has not proved very satisfactory in our hands.

The only really satisfactory method of getting rid of the growths is to remove them by operation. In the great majority of cases this should be accomplished by the intra-laryngeal operation under the guidance of a laryngoscopic mirror held in the left hand, the right hand being free for manipulating the instruments. We need not enter into any detailed descriptions of the methods of procedure to be adopted; their technique can only be acquired by long and careful practice, and without this the intra-laryngeal removal of growths is attended with grave risks of serious injury to the healthy structures. The use of a 20 per cent solution of cocaine or eucaine hydrochloride usually does away with the necessity for long and repeated introduction of instruments in order to inure the patient's larynx to the interference of foreign bodies. In some cases, however, in which the difficulty is not so much due to local irritability as to nervousness, some practice may be unavoidable before an attempt can be safely made to remove the new growth. In our opinion Mackenzie's cutting forceps, or the forceps devised by one of us (W. W.), with changing ends cutting or grasping in any direction (Fig. 30), are the most generally serviceable instruments, but in special cases others, such as Moritz Schmidt's very slender forceps, may be preferable. Dundas Grant's safety forceps are very well suited for growths on the free edge of the vocal cords about their middle thirds. In some cases the galvano-cautery, Löri's cutting catheters, the laryngeal snare, or cutting curettes may be better adapted for dealing with the neoplasm; in fact the choice of instrument will depend almost as much on the tastes and habits of the operator as on the shape of the growth.

Often great difficulties have to be overcome before the growths are finally eradicated, and some cases even now baffle the most skilful operator for a long time; yet by patience and perseverance a very satisfactory result may be confidently anticipated in the overwhelming majority of cases.

The recent ingenious modification by Dr. Horsford of an older instrument intended to pass a thread through the epiglottis and get this out of the way seems well adapted to facilitate and simplify intra-laryngeal removal of benign growths.

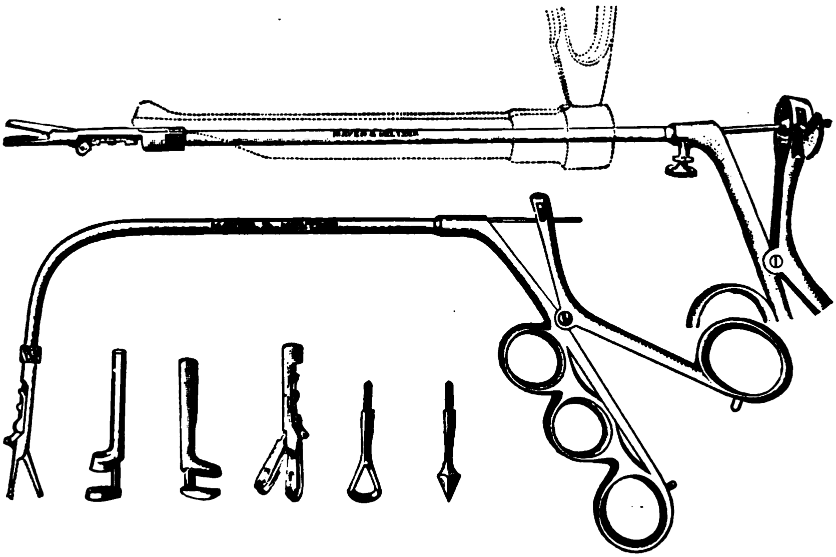

FIG. 30.—Watson Williams's universal forceps for use either by the direct method through Killian's or Kirstein's tube, or by the indirect method.

Should the growth be very large, and should there be a risk of its impaction in the glottis, and of suffocation, prophylactic tracheotomy should be considered, even if removal of the tumour by intra-laryngeal operation may be fairly anticipated; or at any rate during the time of this danger the patient should be placed where tracheotomy could be performed in an emergency. Such measures, of course, are only required in very exceptional cases.

In dealing with multiple papilloma in young children we have some special difficulties to face, both in regard to diagnosis and treatment: in diagnosis from the obstacles to a satisfactory laryngoscopic examination, though there are surprising exceptions to this rule; and in treatment from the clouding of the mirror by mucus, even when a general anaesthetic is used. The ordinary intra-laryngeal method has succeeded in but very few of these cases; and thyrotomy, in addition to its added risks of permanent impairment of the voice, has given no immunity against their recurrence in a great many instances, in spite of apparently very thorough removal of the growths. Mr. Lambert Lack has found it comparatively easy to obtain a view of the larynx in young children by passing the tip of the left forefinger into the right pyriform sinus and hooking forward the hyoid bone, and with it the epiglottis and base of the tongue. Instead of the finger a long tongue-depressor may be used, with the distal end bent down abruptly to the extent of half an inch, or the fish-tailed tongue depressor of Kirstein. Mr. Scanes Spicer some years ago introduced a method which combines general chloroform narcosis with frequently repeated local moppings of the pharynx and larynx of the patient until all secretion is thereby arrested, when he finds it possible to examine the patient laryngoscopically, and if necessary, to proceed at once with the removal of the new growths, should such be found. This method has proved very successful in quite a number of cases.

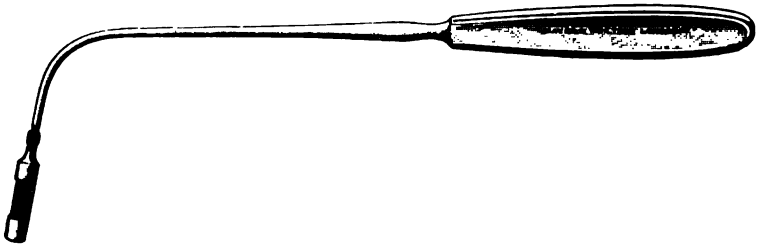

FIG. 31.—Löri's laryngeal curette.

In young children, if there be no respiratory embarrassment, removal of the growths may be deferred with advantage: first, because of the tendency to recurrence; and secondly, of the special difficulties in operating. Should there be any dyspnoea, tracheotomy should be performed, and the removal of the growths themselves postponed to a later period of life, when the child may have gained self-control enough to allow of intra-laryngeal interference.

We are not in favour of intubation, which has been recommended in these circumstances in order to do away with the dyspnoea, and to promote absorption of the new growths. In the first place, no authenticated case is known to us in which absorption of the growths has resulted from this method; and secondly, there must be serious risk of detaching fragments, and of pushing them down into the lower air-passages. On the other hand, removal by means of Löri's catheter-like instrument with windows, the edges of which are cutting, and which is a safe instrument, has sometimes proved very successful. Again, Dundas

Grant's safety laryngeal forceps, or the guarded cutting curette end of the universal laryngeal forceps introduced by one of us (W. W.), are well suited for the purpose, if the indirect method, namely, with the use of the laryngoscope mirror, is adopted.

Of late years the direct method of removal through Kirstein's tube-spatula, or through Killian's model of Kirstein's fish-tailed laryngeal tube, has rendered the growths more completely accessible for removal, and in children for whom a general anaesthetic is necessary, renders successful extirpation much simpler. The child is placed in the dorsal position, anaesthetised, and the parts cocained: the end of the fish-tailed spatula is then passed down over the base of the tongue till it rests against the median glosso-epiglottic fold, behind the epiglottis, and by tilting the distal end the epiglottis is raised so that the interior of the larynx appears in view. With Killian's or Paterson's forceps or Watson Williams's direct action forceps, the growth can then be picked off, the blood draining into the naso-pharynx.

When, from the peculiar nature of the case, external operation is necessary, there are two alternatives: (i.) Thyrotomy; (ii.) Subhyoid pharyngotomy. For subglottic growths, producing respiratory embarrassment, thyrotomy is sometimes unavoidable; but the cases which cannot be dealt with by the natural passages are very few; and it has justly been laid down as a rule that a radical external operation in a case of benign laryngeal growth ought never to be undertaken unless an experienced laryngologist has failed to remove it by intra-laryngeal methods.

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Pachydermia Laryngis—Singers' Nodes.—The term pachydermia laryngis was originally applied by Virchow to circumscribed or diffuse thickening of the epithelium and subepithelial tissue of the vocal cords and other parts of the larynx covered by pavement epithelium, and of the ventricular bands.

Causes.—The affection generally occurs in men between the ages of thirty-five and sixty. Amongst its immediate causes are chronic alcoholism and excessive tobacco smoking; it is especially prone to occur in those who subject the voice to prolonged strain. In not a few cases, however, no definite cause can be assigned for its appearance. Dr. Jobson Horne, whilst recognising the influence of smoking and alcoholism and of faulty voice-production as possible exciting causes, considered that the essential causes were rather to be found in the organism as a whole than in the larynx itself. Syphilis and tuberculosis too are amongst the more important exciting factors; he regarded the pachydermia laryngis found in some cases in association with chronic interstitial nephritis as part of a general fibrosis.

Pathology.—In addition to the thickening and cornification of the epithelium, the subepithelial connective tissue is thickened and sends papilliform processes into the epithelial layer. Inflammatory round-celled infiltration appears, but there is always a distinct line of demarcation between the epithelium and the connective tissue. The local thickening is often surrounded by more or less diffuse congestion and inflammatory thickening. Virchow described the cases due to syphilitic or tuberculous laryngeal disease as secondary or symptomatic forms of pachydermia; these varieties, however, need not be noticed here. Every degree of thickening may occur, from the slightest elevation, due to the heaping up of a few epithelial cells, to a well-defined lenticular tumid outgrowth a quarter of an inch or more in length.

Symptoms.—Often no symptoms are noticeable; but hoarseness and discomfort, slight pain, and considerable impairment in the compass, strength, and quality of the singing voice may be produced.

Objectively the thickening is generally observed on the vocal processes, or inter-arytaenoid fold, on one or both sides of the larynx (Fig. 1, Plate IX.). If bilateral, the wart-like growths are symmetrically placed, and, in the later stages, there is invariably a crateriform depression or pouch at the summit of one side which appears to fit into a corresponding elevation on the other; thus apposition of the local cords is retained and the voice is preserved. This unilateral crateriform depression was explained by Fränkel as probably the result of pressure by the opposite elevation, and not of the firmer fixation of the mucous membrane to the connective tissue at this spot, as Virchow believed; if the latter view were correct, the depression would not be invariably unilateral. Dr. Jobson Horne's observations lend support to Virchow's views; he found no evidence of pressure or attrition, but on the other hand the mucous membrane was more intimately adherent to the underlying cartilage. He further observed that the vocal cords came to lie on different planes, and thus the lines of hyperplasia interdigitate. Diffuse chronic laryngitis, chronic inflammation of the mucous membrane of the larynx, and even chronic adhesive perichondritis may coexist with the pachydermial affection, and sometimes render the diagnosis less easy.

Chorditis tuberosa, or "singers' nodule," or "teachers' node," is a clinical variety of pachydermia. A peculiar small poppy-seed-like growth appears on the upper surface and free border of one or both vocal cords, generally about the junction of the anterior third with the posterior two-thirds of its length. Possibly the tendency in them to occur at this particular spot may be that in singing there is a nodal point here which is subject to continual attrition. These nodules are the consequence of over-use or wrong use of the voice; they interfere particularly with the production of the notes of the upper register, and are most commonly seen in sopranos and tenors (Fig. 2, Plate VII.).

The nodes are merely local hypertrophies of the epithelium and subepithelial connective tissue of the vocal cord, and are usually very hard and consistent. If considerable in size, a small blood-vessel may often be seen coursing over the surface, and a circumscribed hyperaemia of the immediate neighbourhood is frequently present.

The diagnosis rarely presents much difficulty unless the pachydermia be complicated by chronic laryngitis or perichondritis. The crateriform depression above referred to is pathognomonic of the affection, and, in our experience, the mobility of the vocal cords is always unimpaired: impaired abduction of the vocal cords, however, has been described. Early malignant disease of the vocal cord may simulate pachydermia, but in this case impaired mobility of the vocal cord would almost certainly be present; and bilateral affection of the cords favours the diagnosis of pachydermia. In doubtful cases examination of a removed fragment may be possible; but only positive evidence of cancer would be of any value, and the failure to discover anything characteristic of malignant disease should have no weight in cases in which the clinical appearances were indicative of malignancy.

A difficulty may arise in distinguishing between simple or idiopathic pachydermia and the epithelial thickenings and outgrowths that sometimes spring from syphilitic deposits; especially as these forms are but little affected by antisyphilitic treatment. Similarly tuberculous deposits in the inter-arytaenoid fold may give rise to difficulty in diagnosis, if bacilli cannot be found in the sputum and if the pulmonary conditions are indefinite.

The prognosis as regards life and function is—in our own experience—invariably favourable, although we are aware that transition from a pachydermia into a malignant growth has been described; but the affection resists treatment and is very apt to recur.

Treatment.—In our experience the patient practically always gets well under prolonged vocal rest and the steady use of iodide of potassium; especially if any contributory causes, such as smoking and alcoholism, are corrected. Attempts at removal by operation are apt to set up

PLATE X

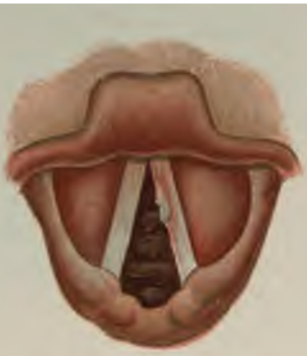

Fig. 1.

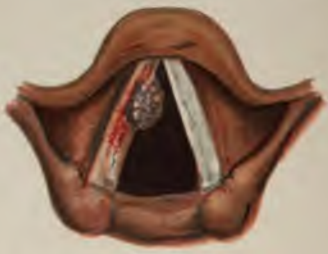

Fig. 2.

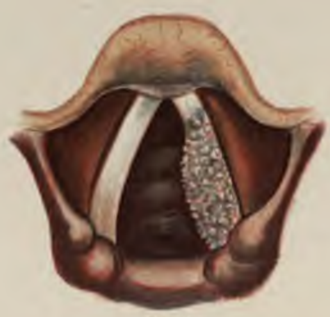

Fig. 3.

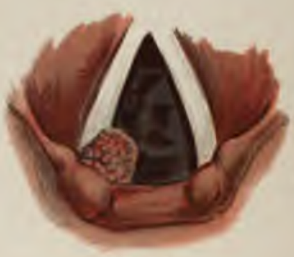

Fig. 4.

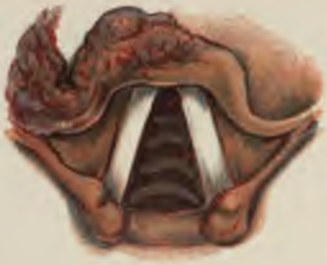

Fig. 5.

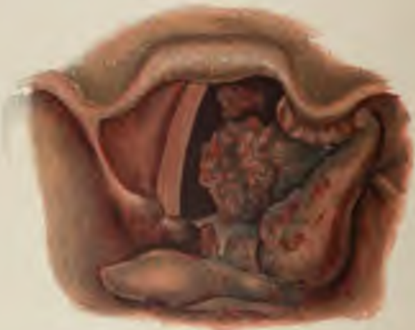

Fig. 6.

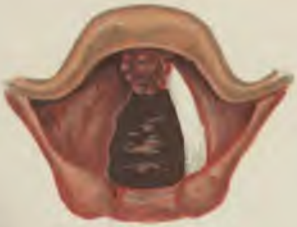

Fig. 7.

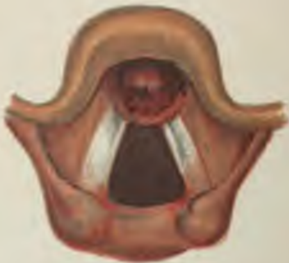

Fig. 8.

PLATE X.

MALIGNANT NEOPLASMS OF THE LARYNX, AND BLOOD-CLOT RESEMBLING OEDEMATOUS FIBROMA.

Figs. 1 to 4. Intrinsic growths.

Fig. 1. Epithelioma of the left vocal cord. (Middlemass Hunt.)

,, 2. Epithelioma of the right vocal cord. (Case of F. S.) Note the hyperaemic zone around the growth.

,, 3. Epithelioma of the left vocal cord, with the snow-white, mown-grass aspect so suggestive of malignancy. (Case of F. S.)

,, 4. A growth thought to be malignant, but in which the histological examination was doubtful. (F. S.)

,, 5 and 6. Extrinsic carcinoma of the larynx. (Cases of P. W. W.)

Fig. 6 is the *laryngoscopic* appearance of the specimen of the larynx illustrated in the text (Fig. 32).

,, 7. A malignant growth has been removed from the right vocal cord. The growth seen in the anterior commissure was not, as it at first suggests, a recurrence, but a granuloma. (Case of F. S.)

,, 8. A blood-clot in the anterior commissure resembling an oedematous fibroma. (Case of F. S.)

perichondritis; electrolysis, under cocaine, with bipolar instruments, has been recommended by Chiari.

Singers' nodules also frequently disappear under the influence of prolonged vocal rest; but they may require operative removal by means of the galvano-cautery, or by means of very delicate forceps. Operation in these cases, ought, of course, only to be resorted to in cases which do not respond to any other form of treatment, as the slightest injury unintentionally done to the cord in the course of the operation may lead to lasting impairment of the singing voice.

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MALIGNANT DISEASE OF THE LARYNX.—**Etiology.**—The causes of malignant growths in the larynx are as obscure as are the causes of malignant growths in other parts of the body. Heredity, excessive use of the voice, and long-continued local irritation are commonly held to have some influence in their production; but the experience of one of us (F. S.), who has had the opportunity of seeing an unusually large number of cases of malignant disease of the larynx, and has paid special attention to these factors, lends no support to these surmises. As a matter of fact, it is hardly ever possible to assign the cause of the occurrence of malignant disease of the larynx.

Men are certainly much more frequently affected than women, and the disease belongs especially to late adult life, being seldom met with before forty. The thirty years of life between forty and seventy supply the overwhelming proportion of all cases of malignant disease of the larynx coming under observation; and of these thirty years by far the largest place is taken by the decade between fifty and sixty. It must be stated, however, that a comparatively large number is met with in the decade from forty to fifty; that is to say, in that portion of life in which innocent growths also are not uncommon, and in which the differential diagnosis between benign and malignant growths, particularly in the earliest stages, is sometimes one of the greatest possible difficulty. Carcinoma is exceedingly rare in early life, but Chiari shewed a girl aged sixteen years at the International Laryngological Congress at Vienna in 1908 with undoubted squamous-celled carcinoma of the larynx; the diagnosis had been confirmed by a histological examination of fragments of the growth removed intra-laryngeally on two occasions.

Pathology.—Both carcinoma and sarcoma occur in the larynx; and of these the former is met with far more frequently than is generally believed, whilst the latter is very rare. The description of both may well be combined; for the symptomatology, diagnosis, prognosis, and treatment of the two forms are so much alike that to describe them separately would entail useless repetition. Carcinoma of the larynx is almost always either primary, or arises by direct extension from neighbouring structures; it almost never is due to metastasis or secondary infection. This immunity is due to the arrangement of the lymphatics of the interior of the larynx, which are very richly developed, but form a network of their own without much anastomosis with the lymphatics of neighbouring structures; they empty themselves into two small glands on each side, one beneath the greater cornu of the hyoid bone, the other at the side of the trachea. This peculiar arrangement of lymphatics is a point of the greatest clinical importance, for it explains, in the first place, why the larynx hardly ever becomes affected secondarily in carcinoma of other parts of the body; and, secondly, why malignant disease occurring in the interior of the larynx tends to remain localised for a long time without affecting neighbouring lymphatic glands of the neck and other tissues: sometimes, indeed, secondary glandular enlargement may be absent to the very end. Consequently, following Krishaber, we shall subdivide cases of malignant disease of the larynx into the *extrinsic variety*, affecting the epiglottis, arytaeno-epiglottidean folds, arytaenoid regions, inter-arytaenoid fold, and the posterior surface of the cricoid plate, and into the *intrinsic variety*, including the growths originating from the vocal cords, the ventricular bands, the ventricles of Morgagni, and the subglottic growths within the borders of the larynx proper.

In the great majority of cases the cancerous growths appear in the form of squamous-celled carcinoma; much more rarely we meet with medullary and hard carcinoma. Columnar-celled carcinoma and villous cancer have each once been seen by one of us (F. S.), and spheroidal-celled or glandular carcinoma has been described by several observers.

Sarcoma occurs in the round-celled and spindle-celled forms, and additionally in combination with other forms of growths, as lymphosarcoma, myxosarcoma, fibrosarcoma. The histological characters of the varieties of malignant growths in the larynx differ in no essential characters from malignant growths generally.

As regards the situation of the growth, intrinsic cases are, in our experience, met with more frequently than extrinsic. Amongst the extrinsic forms, malignant disease of the posterior surface of the cricoid cartilage seems to be by far the most frequent; whilst in the intrinsic forms, so far as can be made out, malignant disease of the vocal cords heads the list by a long way. It is very curious and interesting, that whilst cancer of the larynx is much rarer in women than in men, extrinsic cancer, particularly that form which starts from the cricoid plate, is much more common in women than in men.

In Semon's series of 212 cases, presently to be referred to, there were 124 cases of intrinsic and 53 extrinsic cancer in men, against 12 cases of intrinsic and 23 extrinsic in women. The reasons for this peculiar and striking difference are quite unknown. In a very large proportion of cases the exact starting-point cannot be ascertained with certainty; only too often patients do not seek the advice of the specialist until the disease is already in an advanced stage. These statements are well exemplified by a series of 212 cases seen between 1878 and 1906 by one of us (F. S.) in private practice. Of these 136 were intrinsic and 76 extrinsic or mixed. In the first 103 of these cases, excluding 10 in which the disease was mixed, the points of origin were as follows:—

EXTRINSIC	
Epiglottis	8
Arytaeno-epiglottic ligament (probably)	5
Inter-arytaenoid fold (probably)	6
Posterior surface of cricoid cartilage	19
Total	38

INTRINSIC	
Vocal cords	15
Ventricular bands	3
Ventricle of Morgagni	2
Not to be made out with certainty	35
Total	55

Symptoms.—These vary greatly, not only in different stages of the disease, but also with the situation of the growth; whilst there is comparatively little difficulty in diagnosing the real nature of the affection when in an advanced stage, it is of the greatest importance from the therapeutical standpoint duly to recognise its earliest manifestations. It is therefore essential that careful attention should be paid to symptoms and to laryngoscopic aspects of the larynx, which at first sight may appear almost trivial.

Hoarseness, in intrinsic cases, is nearly always the earliest and most frequent symptom. Its degree, even in the earliest stages, when but a small tumefaction or projection from the vocal cord is to be seen, often is out of proportion to the size of the neoplasm. This is due to the infiltrating character of malignant growths, in consequence of which the mobility of the affected cord, as a rule, is impaired at an early period also (see Diagnosis). As the disease progresses the hoarseness is changed to complete aphonia; but, on the other hand, the voice may return to some extent for a short time as the growth begins to break down, and thus temporarily the vocal cords are brought better together.

In cancer of the epiglottis the voice may remain normal to the end, except for its often having a curiously thick, guttural timbre, which, to the expert ear, sometimes arouses suspicions as to the nature of the complaint as soon as the patient begins to tell his story; whilst in cases in which the arytaeno-epiglottic folds or the posterior surface of the cricoid cartilage are first attacked, it may remain unaffected for a long time.

Pain may occur either at an early or at an advanced stage; but it is often insignificant, and we have observed cases in which this symptom was almost entirely absent throughout the whole course of the disease. Especially is this the case in the intrinsic variety. If present, it may radiate from the throat to the ear, the irritated fibres of the superior laryngeal nerve transmitting the irritation to the auricular branch of the pneumogastric nerve; yet this irradiation of painful sensation is by no means characteristic of malignant disease. Tenderness on pressure over the affected side of the larynx may often be elicited; and when the growth is considerable the larynx is sometimes found notably broadened in consequence of pressure from within. Pain on swallowing is sometimes observed in epiglottic growths: it is most marked when the disease is situated on the posterior wall of the larynx.

Cough, as a rule, is not a prominent symptom. Very rarely spasm of the larynx is observed at an early period of the disease. Increased salivation from reflex irritation and increased secretion from the mucous glands are generally present, and, in consequence of the odynphagia, the saliva collects, and, in the more advanced stages, may dribble out of the mouth. The secretion is at first frothy; later it is tenacious, semi-purulent, and streaked with blood. When the growth ulcerates, and especially when the perichondrium becomes affected, the secretion is fetid, and a peculiar sickly, foul, musty odour is imparted to the breath. Respiratory obstruction depends on the size and situation of the growth. In the later stages of the intrinsic variety it is usually one of the most prominent symptoms. In the extrinsic variety it may result from the growth, if this be situated on the posterior surface of the cricoid cartilage, gradually destroying the muscular substance of the posterior crico-arytaenoid muscles, and thus producing more or less complete paralysis of the abductors of the vocal cords. In the earlier stages slight haemorrhages are common, and when ulceration of the growth has gone far, considerable haemorrhages may occur.

Cancerous cachexia is sometimes absent throughout; particularly in intrinsic cases, owing to the arrangement of the lymphatics, to which attention has already been drawn: but when the growth has spread to the pharynx the characteristic cachectic aspect is seldom long delayed. In large ulcerating growths, especially when extending into the oesophagus, the constant difficulty in deglutition may result in rapid wasting and loss of strength. As a rule, as soon as the disease invades the pharynx the lymphatic glands beneath the sterno-mastoid and the cervical glands become enlarged, either at one or at both sides of the neck, corresponding to the situation of the primary growth.

Signs.—Malignant disease of the larynx, in its earlier stages, may appear on the vocal cords as (1) a single unilateral congestion; (2) a diffuse infiltrating growth, with a red uneven surface; (3) a white, dirty white, or reddish-grey, broad-based, rarely pedunculated, semicircular or oblong wart, bearing a resemblance to a benign papilloma or fibroma; (4) an uneven fringe-like outgrowth from the cord. On the ventricular bands or arytaeno-epiglottic folds and other parts of the larynx, it may appear as a definite tumour, or as a deep greyish-pink infiltration, with a coarsely mammillated or uneven surface. Epiglottic growths are frequently more of a greyish or whitish pink, and may look almost fibrous in texture, but with an uneven surface. Not rarely the disease originates in the ventricle of Morgagni, when it may simulate a growth from the vocal cord or from the ventricular band. In such cases it is sometimes distinctly pedunculated. Various examples of malignant disease of the larynx, especially in the earlier stages, are illustrated on Plate X.

The disease may progress very slowly indeed at first, so that, even after the detection of a definite "wart," no appreciable alteration in size may have taken place after an interval of three or four months; on the other hand, rapid increase in size and early implication of neighbouring portions of the larynx is the more usual course, and points to malignity, especially if a growth which originally occupied the middle or posterior part of a vocal cord extends towards the arytaenoid cartilages and posterior wall of the larynx. As the growth progresses it tends to ulcerate, at first superficially; and it readily bleeds. But deep ulceration is seldom long delayed: the floor of the ulcer is then covered with foul greyish mucopus and debris tinged with blood. As the growth and ulceration extend, the cartilages often become involved; and secondary perichondritis, which may proceed to suppuration and exfoliation of cartilages, not infrequently complicates the disease and may quite obscure its objective symptoms.

Sarcoma generally originates as an ill-defined, infiltrating growth, the primary seat of which often cannot be ascertained. It may spring from any part of the larynx. The growth, if defined, is smooth, globular, and often semi-translucent, but it may take the form of a greyish-pink infiltrating tumefaction with smooth but uneven surface. The rapidity with which it extends varies greatly in different cases.

The patient very rarely lives more than three years after the appearance of malignant disease of the larynx, if it be left to run its ordinary course; usually indeed the duration of life is considerably shorter. With advancing weakness and emaciation, and sometimes in a general cachectic condition, the patient sinks and dies; in many cases he is carried off by some intercurrent affection, such as bronchitis and congestion of the lungs, or by a "foreign-body pneumonia," due to the escape of particles of food or secretion through the distorted glottis into the lower air-passages. Sometimes he dies from sudden heart failure; in other cases the secondarily affected cervical glands break down, and add to the sadness of the closing scene.

Diagnosis.—The chief points which should attract our attention in cases of early malignant disease of the larynx are the age of the patient, the symptoms, especially that of hoarseness coming on without an obvious cause, the laryngoscopic appearances, the absence of general symptoms pointing to pulmonary tuberculosis, syphilis, or gout (all of which, of course, do not *exclude* a concomitant cancer of the larynx), and—where possible—the histological character of portions of any growth removed for diagnostic purposes. The affections with which laryngeal carcinoma is most likely to be confounded are inflammatory diseases, including the formation of blood-clots, which, as stated in a previous section, may in a surprising manner simulate malignant growths; syphilis, tuberculosis, lupus, laryngeal palsies, gout, benign growths, pachydermia laryngis, and perichondritis.

In those cases in which malignant disease first manifests itself as a diffuse hyperaemia, it usually is distinguished from chronic laryngitis by its being unilateral; this character in itself would suggest to an experienced laryngologist the probability—though by no means certainty—of the beginning of some serious affection, such as carcinoma, tuberculosis, or syphilis. In exceptional cases, however, what seemed to be an ordinary chronic laryngitis affecting both vocal cords, may ultimately turn out to have been the starting-point of malignant mischief. A case of this kind has been described by one of us (F. S.).

After a time, in most cases increasing heaviness in the movements of the diseased vocal cord will be observed, which, taken in conjunction with the accompanying circumstances, the age of the patient, abnormal sensations of pain, and sensitiveness to pressure on the affected side, is a very suggestive sign. In those cases in which this heaviness in the movement of the cord is present, it is a very valuable sign in distinguishing malignant growths from benign neoplasms and pachydermia verrucosa. It cannot, however, be too strongly stated that this heaviness is not *invariably* present, and that its absence must not be interpreted as a proof against the existence of malignant disease. Sometimes this sluggishness of movement is seen at a very early stage of the disease, when the growth may be no larger than a pea. If this sign be absent from a case in which almost the entire vocal cord appears to be embedded in a papilloma-like mass, and in which age and other symptoms point towards malignancy, it is well to remember that the growth may have arisen from the ventricle of Morgagni; an origin which would explain the absence of this valuable sign. The growth, whether pedunculated or sessile, is generally surrounded by a circumscribed, diffused, dirty pink hyperaemia, which is often in striking contrast with the whiteness of the remainder of the cord and of the healthy one (Fig. 2, Plate X.). Such growths may start from any part of the vocal cord, but—in contrast to the usual seat of benign papilloma—are very apt to originate from the middle or posterior third of the vocal cord, a site which when seen in patients over fifty years of age should always suggest grave suspicions (Fig. 4, Plate X.). In colour they vary from an almost chalky white to a pink or dusky red; and their surface may be either smooth or granular, or mammillated. In a case observed by one of us (F. S.), the surface, its white colour apart, could best be compared to a newly-cut grass lawn (Fig. 3, Plate X.); in another the appearances were those of a pedunculated angioma.

In other cases, again, the neoplasm is almost indistinguishable from a benign papilloma; yet a particularly fine branching of the individual papillae, or the embedding of an entire vocal cord in a greyish white or reddish papilloma-like mass, or the appearance of a fringy papillomatous edge along its entire length, especially if one or more of these signs be observed in an elderly patient, will put the experienced observer on his guard. If, after removal of an apparently benign growth, rapid recurrence take place—especially if the recurring neoplasm be covered with an abundant growth of vegetations—or if the wound left by the removal of the whole or a portion of the growth fail to heal and present a sloughing, unhealthy aspect, malignancy should be strongly suspected. When the growth is larger in size the diagnosis is, of course, much easier. Large malignant growths would be distinguished from benign ones by their irregular form, early ulceration, and tendency to bleed, whilst, of course, any enlargement of the cervical lymphatic glands would be noted, and in a patient of advanced years would point strongly to malignant disease.

To recapitulate; it is the early cases without distinctive signs that give rise to difficulties that make the expert pause before committing himself to a definite opinion. In differentiating between tuberculosis, syphilis, and malignant diseases, the following conditions call for special consideration. (1) *Congestion of the vocal cords:* (*a*) Bilateral. Although bilateral congestion is in the majority of cases due to catarrhal influences or over-use of the voice, it may occur in syphilis and tuberculosis, and in both it is often only the obstinacy of the congestion and its refractoriness to treatment which lead the observer to the conclusion that he has something more serious than catarrh to deal with; but the apparent bilateral catarrh may be, as we have already remarked, the precursor of malignant disease. (*b*) Unilateral. Although a marked unilateral congestion of one vocal cord only is highly significant of either tuberculosis, syphilis, or malignant disease, it is not invariably so, and one must not consider this an infallible sign of something more serious than simple catarrh.

(2) *Tumours:* (*a*) Tuberculous tumours may arise at any time of life, rare as they are, and may start from any part of the larynx. They are covered by normal mucous membrane, with a smooth, somewhat granular surface, of a grey, yellowish-reddish, whitish colour, and are usually roundish in form or almost globular. The difficulty of distinguishing between tuberculous and malignant growths is the greater, because tuberculous tumours occur in cases in which there may be no demonstrable pulmonary lesion, and no cough, and also because they are often so smooth and rounded that it is exceedingly difficult to remove a fragment for histological examination; especially will great difficulty in differentiating a malignant growth occur, if in an elderly patient a tuberculous tumour arises in such situations as the ventricle of Morgagni, the posterior wall of the larynx, or in the subglottic cavity below the anterior commissure (*vide* p. 199). (*b*) Again, gumma in the pre-ulcerative stage might be confounded with either tuberculous or malignant growths, but the diminution of the growth under iodide of potassium, or, where

this has not been given, the rapid breaking down of the swelling with deep crateriform ulceration, soons clears up the difficulty (*vide* p. 214).

Diffuse infiltrations afford the greatest difficulty of all in diagnosis; for either of the diseases may manifest itself in this form, particularly when, in the absence of any general symptoms pointing to the true nature of this disease, the diagnosis has to be made from the laryngoscopic aspect of a diffuse infiltration alone, occupying either the posterior wall of the larynx or one of its sides, and being in some cases still covered with normal mucous membrane, or in others already superficially ulcerated. Nor must it be forgotten that a syphilitic or tuberculous patient may develop laryngeal cancer, with or without concomitant tuberculous or syphilitic laryngeal lesions. Malignant growths undoubtedly sometimes originate from parts of the larynx other than the vocal cords, and in such cases the general submucous infiltration, gradually invading various laryngeal structures, might be mistaken for a sign of perichondritis from causes other than malignant.

The posterior thirds of the vocal cords are rarely, and the inter-arytaenoid fold is practically never the seat of benign growths, whilst these are the regions in which Virchow's pachydermia verrucosa is most frequently found. The free movements of the vocal cord in the last-named disease, the relatively less pronounced hoarseness, and a history of chronic alcoholism are strong points in the diagnosis of these excrescences which, moreover, particularly in more advanced stages, tend to become bilateral, when a crateriform depression on the vocal process of one of the cords, into which the summit of the tumefaction of the opposite vocal process fits, often is eminently characteristic.

It is a very good practical rule, in all cases of suspected malignant disease, to administer iodide of potassium for a while, even when there is no history of syphilis, in 10-grain doses at first, rapidly increased to 30 grains, three times a day. But we would again point out that mere subjective improvement after the administration of this drug is not to be trusted; for patients undoubtedly suffering from malignant disease often declare themselves better after taking the iodide, which acts as a resorbent upon the area of congestion around the new growth; we must therefore be guided solely by the objective changes in the size of the growth or infiltration itself.

In a few rare cases the appearances of an infiltrating malignant growth are so closely simulated by chronic infective inflammatory processes, the exact nature of which has not yet been definitely settled, that errors are almost unavoidable. One of us (F. S.) has twice met instances of this kind. The only possibility of distinguishing between malignant and such infective infiltrations would seem to consist in intra-laryngeal removal and microscopic examination of a fragment of the suspected tumefactions; unfortunately, however, they usually are of so diffuse character, that it may be extremely difficult, if not impossible, to remove intra-laryngeally a piece sufficiently large to enable the histologist to form a definite opinion thereon.

This brings us, finally, to the question of the value of a microscopic examination of a removed fragment of a suspected growth. When this reveals to a competent pathologist positive and unmistakable evidence of the malignancy of the growth—as in cases of squamous-celled carcinoma (epithelioma)—there is, of course, no room for doubt as to its character; but we cannot too strongly emphasise the importance of remembering that a mere negative verdict of the pathologist must not set aside clinical apprehensions otherwise well founded. The possibility that the growth is of a mixed character, or a papillomatous surface growing from a malignant base, ought always to be remembered. In short, microscopic examination of the fragments removed intra-laryngeally is a valuable but not infallible aid to diagnosis. Every portion of the removed fragment should be cut into sections and each one carefully inspected; and if the examination reveal no character of malignancy a further and deeper portion should be removed if the clinical appearances suggest any suspicion as to its nature. The apprehensions, repeatedly expressed by Dr. John Mackenzie of Baltimore, as to mischief resulting from the intra-laryngeal removal of fragments for the purpose of microscopic examination, we do not share. We would, however, strongly advise, in the practitioner's interest, that such probatory removal should only be resorted to if the patient consents beforehand that radical operation should be performed forthwith, if the microscope should reveal the malignant nature of the new growth. However, there are but too many cases in which the disease appears in the form of a general smooth infiltration from which it is almost impossible, intra-laryngeally, to remove portions for microscopic investigation. In such cases the clinical observer must have the courage to form a definite diagnosis from clinical signs only.

The **prognosis** varies enormously according to (*a*) the original situation of the growth; (*b*) the stage and extent of the disease at the time at which the patient comes under observation; (*c*) the patient's age and general health. A small intrinsic malignant growth in an otherwise healthy middle-aged patient allows of a much better prognosis (provided that immediate radical operation be consented to) than was considered possible some years ago, whilst the outlook in cases of extrinsic or of very extensive intrinsic growth in very old patients, or in those whose general health has suffered from other causes, is still extremely grave.

The **treatment** may be considered under two headings, radical and palliative. The radical treatment of malignant disease of the larynx may be said to have undergone a complete transformation within the last twenty years; for whereas attempts to extirpate the disease were so disastrous that they were rarely considered justifiable after von Bruns's statistics of 1879, the experiences of Mr. Butlin and of one of us (F. S.), who have been fortunate in having exceptional opportunities of treating laryngeal growths surgically, have yielded results which, when we remember the inevitable and speedy end of all such neoplasms when left to run their natural course, are most gratifying.

Before considering external operations, it may be stated that B.

Fränkel of Berlin, who in 1887 warmly, though cautiously, recommended the intra-laryngeal method, still adheres to his views; that he is supported in this by Jurasz, Bresgen, Krieg, Baginsky, Finder, and a few other laryngologists; and that a small number of cases has been reported in which, after laryngoscopic operation, a cure has been obtained. It is, however, held by most experienced laryngolists, in view of the infiltrating character of malignant growths, and of the fact established years ago by one of us (F. S.), and since corroborated by many observers, namely, that when the larynx is opened the disease is frequently found to be much more advanced than was apparent on laryngoscopic examination, that the advantages which are claimed for the intra-laryngeal method are far outweighed by the risks of promoting a more rapid growth of the neoplasm and by missing the favourable time for external operation, and we cannot, therefore, recommend the intra-laryngeal method in malignant disease of the larynx. The actual size and extent of a growth as compared with the laryngoscopic appearances are well exemplified by Fig. 32, and Fig. 6, Plate X., respectively.

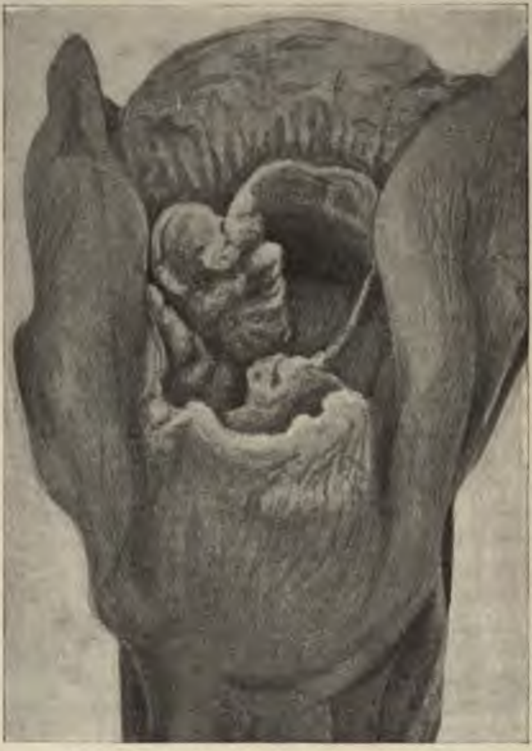

FIG. 32.—Extrinsic malignant diseases of the larynx.

On the other hand, the results of thyrotomy, when performed in the proper class of cases and at the right time, speak for themselves. Out of 33 patients on whom thyrotomy was performed by one of us (F. S.), for presumably intrinsic malignant growths, in 8 the diagnosis was either found to have been at fault or remained doubtful. Twice the disease proved to be tuberculous. Twice the tumefactions were stated by Mr. Shattock to have been of the nature of an infective inflammation. In two cases the diagnosis between fibrosarcoma and innocent growth remained doubtful. In 1 case it still is an open question whether it was of an epitheliomatous or papillomatous character, and in 1 it was subsequently proved to be an innocent papilloma. But, excluding all doubtful cases, there remain 25 cases microscopically verified as undoubted malignant growths; of the 25 patients 1 died and 24 recovered. In 4 cases recurrence took place, but in 3 only of these was the recurrence local, the patient in the fourth case dying from cancer of the liver five years after the operation, whilst the larynx and cervical glands remained perfectly free. Of the three cases in which local recurrence took place, in the first the recurrence was observed nine months after the operation, and the patient died another nine months later, a second operation having been declined. In the second case, the recurrence took place four months after the operation; hemi-laryngectomy and subsequent total laryngectomy were performed, but the patient succumbed to the latter. In the third case, recurrence took place eleven months after the operation; hemi-laryngectomy was performed, and the patient continues to do well nearly 1½ years now after the last-named operation (F. S.). Deducting from the 24 cases in which recovery took place the 2 first cases just named, there remain 22 cases with genuine local recurrence in 3 or 13·6 per cent of recurrences. This gross result becomes even more important when the general question of recurrence is considered. Hitherto it has been almost universally believed that, even if radical operations in malignant disease be at first successful, a recurrence of the disease within a comparatively short time is an almost unavoidable contingency. Now subtracting, from the 25 cases of operation by thyrotomy for undoubted malignant disease of the larynx, 1 case in which death occurred from the operation, and 3 cases in which local recurrence took place within the first year after the operation, but including, on the other hand, 1 case of incomplete operation, in which, after a second thyrotomy, a lasting cure was effected, we have 21 cases of lasting cures remaining, namely, exactly 84 per cent. If, however, the case be deducted in which five years after operation the patient died from cancer of the liver, 20 out of 25 cases, or 80 per cent of lasting cures, would be obtained, a result which, we think, favourably compares with the results of radical operations for cancer in any part of the body.

The duration of the cure in each individual case from the date of the operation to 31st December 1906, has been as follows:—

1 case	over 15 years.
4 cases	between 10 and 15 years.
4 „	between 5 and 10 years.
2 „	over 4 years.
3 „	over 3 years.
2 „	over 2 years.
1 case	just 2 years.
1 „	1 year 10 months.
1 patient died 5 years after operation from pulmonary embolism.[1]	
1 patient died 4 years after operation from pneumonia.[1]	
20	

The vocal results are equally satisfactory; voice surprisingly good in 11 cases, good or fair in 5, weak or reduced to a whisper in 4. In not a few instances both vocal cords had to be removed.

The Operation.—Radical operation having been decided upon in a case which from its laryngoscopic examination justifies the hope that thyrotomy, with the removal of soft parts, and possibly with resection of some portion of the cartilages, will be sufficient, tracheotomy is first performed, and a Hahn's aseptic compressed sponge-cannula is introduced into the trachea. Fully ten minutes should now be allowed to elapse before the larynx is opened, in order to give the sponge full time to expand and to occlude the lower air-passages hermetically, so as to prevent the entry of blood when subsequently the larynx is opened and the operation in its interior performed. Undue hurry in proceeding with the actual operation immediately after the performance of tracheotomy, whereby, after the opening of the larynx, blood may be allowed to enter into the lower air-passages, has probably had a good deal to do with the pulmonary and bronchial complications so often encountered in this class of operations. The ten minutes having elapsed, and the front part of the thyroid cartilage meanwhile having been laid bare throughout its entire length by means of scalpel and raspatory, it is opened exactly in the middle line, bone-forceps or Waggett's shears being usually necessary for this purpose, as the cartilage is almost always found to be calcified in patients of such an age as are those on whom these operations are performed. On account of this calcification, the inspection of the interior of the larynx and the handling of instruments in it is very often extremely difficult; the sides should be held apart either by two broad retractors or, better still, by two strands of strong silk passed through the anterior part of the lateral wings of the thyroid cartilage after it has been split open. Undue violence, of course, in holding the two halves apart should be strictly avoided. At this stage of the operation it will usually, but by no means always, be necessary to protect the field of operation against mucus and saliva, which are often secreted in excessive quantities from the mouth and pharynx, by plugging the lower part of the pharynx by means of a comparatively large aseptic sponge secured by a long string and introduced through the laryngeal wound into the lower part of the pharynx, which it ought to fill up as completely as possible. The operator will also do well to be provided with a frontal reflector, to be attached to his forehead, and with a good source of light, in order to illuminate the interior of the larynx well before and during the removal of the growth. A little Trouvé's or Klar's electric lamp will also be found to be very useful for the purpose. Finally, before commencing the removal of the new growth, the larynx should be twice mopped out with a 10-20 per cent cocaine solution by means of a soft camel's-hair brush at intervals of a minute or two, in order to contract the capillaries on that side and to prevent parenchymatous bleeding, which otherwise is sure greatly to interfere with, and unduly to prolong, the removal of the growth proper, whilst at the same time it may lead to infected portions being left behind. In an operation in which, on the one hand, it is so absolutely necessary not to leave any fragment of the growth behind, and, on the other hand, not to sacrifice more of the important structures one has to deal with than is absolutely required, everything that facilitates the achievement of this purpose cannot be too carefully considered. Many operators nowadays use a mixture of cocaine and adrenalin for the purpose not only of contracting the capillaries temporarily, but also of making the confines of the growth itself more visible; but after having availed himself of this method twice without observing any marked advantage during, and meeting with more copious bleeding after, the operation, one of us (F. S.) has returned to the application of cocaine alone. The field of operation having been thus prepared, the operator should make two semicircular or elliptical cuts, joining in front and behind through the whole of the soft tissues and down to the perichondrium, round the diseased area, and, of course, not too close to it, *i.e.* at a distance of about half to one inch from the periphery of the growth, so as to be certain that he actually operates in the healthy neighbourhood and leaves no part of the tumour itself behind. Desirable as it is not to take more away from the voice-producing parts than is really necessary, no undue sentimentality in this respect must be allowed to interfere with the thoroughness of the operation—life and absence of recurrence first, voice afterwards! In this connexion it may at once be stated that the operator must be prepared to find the disease considerably more extensive than it had seemed from mere laryngoscopic examination, and that during its course the operation may assume a much larger scope than was originally intended.

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[1] In both these cases recurrence was absolutely excluded.

The area to be removed having thus been circumscribed, the removal ought to be effected by the growth itself being held with dressing forceps and the whole area being cut with curved scissors, the points of which are firmly pressed against the inner aspect of the cartilage, the mucous covering of which is to be removed. Unfortunately, it will be found that it is hardly ever possible to remove the whole of the growth in one piece, and, as a rule, the diseased portions will have to be removed piecemeal. It is during this process that the advantage of first contracting the capillaries by means of the cocaine solution becomes obvious. The entire growth and a part of its healthy neighbourhood—if necessary, including portions of the cartilage—having thus been removed, the base should be firmly scraped with a sharp spoon. In very rare cases only will it be found necessary to apply the galvano-cautery in conclusion. Finally, every source of bleeding having been carefully stopped, and even small bleeding vessels ligatured, the whole of the interior of the wound is dusted with deodorised iodoform, or with iodoform and boracic acid mixed in equal parts. The two halves of the thyroid cartilage are then brought together and stitched to one another by catgut ligatures, the needle being passed through the *outer* part only of the cartilage itself, if this be soft enough to allow of this, or even through the covering perichondrium only, if the cartilage be calcified, and care being taken (*a*) that the two halves of the thyroid cartilage be at exactly the same horizontal level; and (*b*) that the ligatures be not passed through the interior of the larynx itself, where they may cause the formation of troublesome granulomas, giving rise to suspicions of recurrence (Fig. 7, Plate X.). We wish to insist most emphatically that the occurrence of such granulomas after thyrotomy is far from being unusual, and to warn the reader not to regard every tumour appearing in the scar a few weeks or even months after operation as a probable recurrence. In every such case, intra-laryngeal removal and microscopic examination of the suspected growth should be carried out before undertaking a second external operation ; it will usually be found that the little intra-laryngeal operation causes complete and lasting disappearance of the suspected growth. In some few cases it will be found impossible to pass needles through the perichondrium. In such cases the deep muscles above the thyroid cartilage must be stitched together to allow, as far as possible, union of the two halves of the cartilage in their natural position. The sponge-cannula is now immediately removed—a step for which, as well as for other valuable simplifications in the technique of the operation, we are indebted to Mr. Butlin. The whole upper part of the external wound is closed by aseptic horse-hair ligatures, these are also passed, while the patient is still under the influence of the anaesthetic, through the lowest part of the wound but are only tied together loosely, so as to allow free drainage from the lower part of the wound, the whole of which is then covered with cyanide gauze. All being well, these lower sutures are tightened after twenty-four hours. By this means the after-treatment is greatly simplified and curtailed. In cases in which, from the very beginning, more or less extensive resection of the thyroid cartilage, or even extirpation of one half of the larynx, is to be anticipated, the parts of the cartilage to be removed ought to be freed from their perichondrium and from the surrounding soft parts by means of an elevator. In other respects the operation is the same, only more extensive than mere thyrotomy with removal of soft parts. It will not be found necessary in most cases prophylactically to ligature one or several of the laryngeal arteries.

In cases in which the malignant disease either affects the epiglottis or the arytaeno-epiglottidean fold, subhyoid pharyngotomy (after preliminary tracheotomy and insertion of Hahn's tube) appears to be the most useful method of operation, as it is the simplest way of giving access to the diseased parts. The incision should be made horizontally and parallel to the lower border of the hyoid bone, across the neck to an extent of about six centimetres. The operator then dissects carefully downwards, and after exposure of the thyro-hyoid membrane divides this, during which act he must be careful not to cut across the epiglottis close to its base, as this part is not always separated from the ligament by the usual layer of connective tissue, but may be adherent to it. The whole pharynx is thus well exposed to view. The larynx having been pulled forward to some extent out of the wound, the growth can now be freely removed with curved scissors or the knife, the operator, of course, always proceeding in the healthy tissue in its neighbourhood. After free removal ligatures are to be applied to all the vessels, the wound is to be disinfected in the manner above described, and the pharynx is to be stitched off from the external wound by means of horse-hair ligatures; whilst in this class of cases the external wound also may be closed immediately by similar ligatures, fine strands of silk being inserted across to serve the purposes of drainage. As in the case of thyrotomy, the wound is then to be covered with cyanide gauze, and a bandage may be applied.

The after-treatment of cases of radical operation for malignant disease of the larynx up to a recent period was very troublesome, both for the patient and for the operator. The sponge tube having been removed at the end of the operation, and the wound having been treated in the manner above described, the patient is not propped up in bed, but placed in an almost horizontal position on his side, the side which corresponds to the half of the larynx operated upon being lowermost; one pillow only is allowed under his head. By means of this position the tendency of secretion to pass down into the lower air-passages is as much as possible diminished. The external covering of cyanide or iodoform gauze must be as often renewed as it is wetted by secretions, and the employment of an attentive nurse is very essential for this part of the after-treatment. Finally, with regard to feeding, unless the operation be very extensive, it will be found that by employing the simple form of after-treatment just recommended, patients will be enabled to take their food through the mouth, usually even on the first day, so that in most cases the troublesome rectal feeding can be altogether avoided. Some hours after the operation it ought to be seen whether the patient, lying quite flat on the operated side, can drink some sterilised water from a feeder introduced into the dependent side of his mouth without this causing cough and escape of the fluid from the lower part of the wound. Should the attempt be successful, the experiment should be repeated with sterilised milk, and if this, again, passes into the oesophagus and stomach without entering into the air-passages, the patient may be safely fed, though cautiously, of course, from the first by the mouth. On the other hand, if violent coughing and escape of fluid through the external wound occur, rectal feeding must be resorted to, until, on repetition of the experiment, the fluid goes the right way. The upper part of the wound almost always heals by first intention; the lower part may also do so, but more frequently closes by the formation of granulations, which may have to be touched by 2 to 4 per cent nitrate of silver solution once a day if they should become too exuberant. As a rule the patient is able to get up by the fourth to sixth day, and is able to leave before the end of one fortnight from the operation.

It is quite possible that, with further experience, the plan of treatment as sketched above may become still further modified. Indeed, of late years, various suggestions have been made by Grünwald, Paul von Bruns, and others as to dispensing with tracheotomy and general anaesthesia altogether, and as to placing the patient's body in the oblique position with the head hanging down.

The following is a brief description of the technique followed by Professor Paul von Bruns. Instead of general narcosis, half an hour to an hour before the operation an injection of scopolamine and morphine is given, and a solution of novocaine is injected into the line in which afterwards the external incision is to be made, whilst after the larynx has been opened, the mucous membrane is painted with a solution of novocaine and adrenalin, in order to abolish reflex cough and deglutitory movements, and to limit bleeding from the mucous membrane. If no graver haemorrhage is to be feared, previous tracheotomy and introduction of a cannula is avoided. With Kocher he prefers the oblique position of the body, with the head hanging down, in order more securely to prevent the aspiration of blood ; this position also protects against the invasion of the pharyngeal mucus—which is often secreted in large quantities—into the larynx, and which has otherwise to be prevented by plugging of the lower parts of the pharynx. The division of the cartilages extends throughout the larynx into the trachea, in order to give sufficient space. When the cartilaginous halves have been drawn asunder, an incision—if necessary under artificial illumination by means of a small electric lamp—is made all round the tumour down to the perichondrium and close to the cartilage. The bared surfaces are, if necessary, burnt with a galvanocautery, and iodoform is rubbed into them. The introduction of an iodoform-gauze tampon is omitted. Finally, the halves of the thyroid cartilage are brought together, and the upper part of the external wound is closed, whilst the lower part in the region of the trachea remains open, and is only loosely plugged. No cannula is introduced. It will be seen that this technique in many material points differs from that followed by Butlin and Semon, and further experience will shew which of the two gives the best results.

With regard to subhyoid pharyngotomy, we see from Professor Paul von Bruns's latest statistics that of all subhyoid pharyngotomies performed for laryngeal cancer since 1890, a cure lasting from one to three or more years had been obtained in 16 per cent only, that in 20 per cent recurrence took place, and that the mortality of the operation was no less than 40 per cent. Quite apart from the fact that it can be employed in a very limited number only of cases, namely, in those in which the disease is situated near the upper aperture of the larynx, the danger of the operation is so great as to give little encouragement for its adoption, particularly since equally good access to the growth can be obtained by laryngotomy. One of us (F. S.) has performed subhyoid pharyngotomy but once for malignant disease of the pharynx, and has met with the same disappointing experiences as others have done, namely, that the patient died on the fourth day after the operation, probably from sepsis.

Turning now to the question of total laryngectomy, we are glad to state that the former terrible mortality of the operation has, through advance in the technique, been very much reduced of late years, and we do not doubt that a few of the patients who have survived the operation for any length of time have been and are quite content with their lot. We therefore wish to state expressly that we unconditionally admit the legitimacy of an operation which saves, or even only appreciably prolongs, a fraction of lives otherwise irretrievably lost. But total laryngectomy is, beyond the shadow of a doubt, one of the most mutilating operations in surgery. It does away with speech in its ordinary sense, for we have never seen a patient avail himself for any length of time of any of the many ingenious artificial larynges which have been invented and recommended. He rather puts up with the pharyngeal whisper, which in some cases is audible at a distance, particularly when intensified by Gluck's ingenious invention of letting the expiratory blast of air pass over an india-rubber tube one end of which is introduced into the tracheal cannula, whilst the other end is held in front of the patient's mouth. Often enough, however, the patient as a matter of fact only communicates with the outside world in writing. In not a few cases to this loss of speech is added permanent difficulty in swallowing, which compels the patient to take his meals by himself or even to be fed artificially. All this more or less shuts off the patient from the company of his fellow-men, partly from instincts of his own, partly from being driven to observe that he is an object of pity mixed with horror to others. He, therefore, usually shuns society and becomes a recluse. The statistics of Chevalier Jackson, the only modern operator of considerable personal experience, who has tabulated the whole of his results, are worthy of careful note. He states that out of eight total laryngectomies performed by him three were hemi-laryngectomies followed by recurrence and total removal. One of the patients lived seven years after, when he died from cancer of the stomach; another lived three years without recurrence, dying of cerebral haemorrhage; and the third died eight months after from alcoholism. Of the remaining five, recurrence occurred in three within a year, one apparent cure was lost to observation after a year, and one is too recent to record. Out of the three prompt recurrences there was one with metastases in the lungs, liver, and pancreas. Thus, out of eight laryngectomies no absolute ultimate cures could be claimed in the author's own opinion, although three were apparently cured at the end of one year. Gluck, whose experiences have been very large, and who is generally acknowledged to be the protagonist and most successful performer of total laryngectomy, unfortunately has not yet put them together in such a statistical form as to enable the reader to judge the total of his final results.

Prof. von Bruns, after collecting all the total laryngectomies performed since 1890, apportions the results as follows:—Cure lasting more than three years, 10 per cent; cure lasting one to three years, 16·6 per cent; cure lasting less than one year, 28·9 per cent; recurrence, 14·5 per cent; death during operation, 19 per cent.

From consideration of these facts and of what has been said about the kind of after-existence which the great majority of the patients have to lead, it appears to us that the proper course to follow, when the question of total laryngectomy comes up, is to lay an impartial statement of the alternatives of allowing matters to take their course and of having the operation performed before the patient, and in no way to influence his decision.

Concerning the technique of total extirpation of the larynx and its after-treatment, we must refer the reader to the textbooks of surgery.

Palliative Measures.—In cases which are unsuitable for radical operation, or in which this is declined, we have to rely on maintaining the patient's general health and strength by suitable tonic remedies, food, and rest. If swallowing be painful, the food should be soft and bland: it is not well to urge patients to go on taking solid food when the local pain and irritation are increased thereby. Insufflation of anaesthesine or orthoform over the ulcerated surfaces ten to fifteen minutes before meals will, for a time at least, materially diminish the difficulty in swallowing. Later on, feeding through a soft tube introduced through the mouth or through the nose, and, in the last stages, rectal alimentation may be required, or—in cases in which the disease starts from the posterior wall of the larynx and in which dysphagia is an early symptom—the advisability of performing gastrostomy will require consideration if total laryngectomy be declined. When respiratory obstruction supervenes, tracheotomy should be performed. Life may be prolonged a good many months in some cases by this operation, if the latter be not too long postponed; and in many patients there is a considerable improvement in other symptoms besides the dyspnoea. The low operation is preferable to the high, as the growth may spread down so as entirely to surround the tube. When ulceration has occurred, the use of antiseptic appliances containing morphine is called for.

We cannot conclude this article without reiterating the hope expressed by one of us (F. S.) in introducing a discussion on the "Diagnosis and Treatment of Laryngeal Cancer" before the Medical Society of London in 1907, namely, "that every practitioner should endeavour to diagnose the intrinsic variety at so early a time, that thyrotomy will suffice, not only to save the patient, but also to help him to an amenable existence afterwards."

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LARYNGEAL NEUROSES

(I.) MOTOR NEUROSES.—*Introductory Remarks.*—The superior laryngeal nerve through its internal branch supplies sensation to the larynx on each side, and its external branch is the motor nerve to the cricothyroid muscle. The recurrent laryngeal is the motor nerve to all the other intrinsic muscles of the larynx. For many years the generally accepted view had been that the fibres of the recurrent laryngeal nerve were ultimately derived from the spinal accessory nerve through its communication with the vagus before it leaves the cranial cavity. According to more recent experiments of Grabower, Grossmann, and Walter Spencer, however, the recurrent nerve is derived from the nucleus ambiguus of the vagus, and not from the spinal accessory. This question, which has been very keenly discussed for many years, is not yet definitely settled.

The experiments of Semon and Horsley, corroborating Krause's investigations, demonstrated that there is in each cerebral hemisphere a cortical centre for adduction of the vocal cords (as in phonation); and that in the left hemisphere this centre corresponds with the speech centre, which in man lies in the anterior portion of the lower extremity of the ascending frontal convolution. Irritation or stimulation of either centre will produce bilateral adduction of the vocal cords, and, if continued for some length of time, spasm of the glottis; whilst destruction of one centre produces no corresponding paralysis so long as the other is intact. Thus, in motor aphasia the vocal cords are not affected; and a unilateral cortical lesion has never been proved to cause unilateral paralysis of the opposite vocal cord, although some French observers (Garel and Dor, Déjerine) strongly maintain that this is possible; and indeed that it has been observed. It is impossible here to enter more fully upon this hotly debated question; and we must refer those interested in it to a paper published by one of us (F. S.), in which it is fully discussed.

Semon and Horsley found a centre for abduction of the vocal cords in the cat lying close to the border of the olfactory (rhinal) sulcus; no abductor centre was found by these observers in any of the other classes of animals experimented upon, although the existence of such a centre in the cortex was almost certain from their discovery of a spot in each internal capsule, excitation of which gave rise to bilateral abduction of the cords. More recently, however, Dr. Risien Russell has discovered cortical centres for abduction in the dog also, which on unilateral excitation produce bilateral abduction of the cords; this result was obtained when the more powerful adductor movements have been to a certain extent abolished by previous section of the adductor fibres in the recurrent laryngeal nerve of one side. Abduction of the vocal cords was obtained from the anterior composite gyrus just in front of and below the adductor centre, and therefore a little in front of and below the anterior extremity of the coronal sulcus.

In further exploring the cortex Dr. Risien Russell found that on the anterior composite gyrus, below the abductor centre, there exists a focus, excitation of which results in what is described as a clonic adductor effect on the cords; in this action the cords were first brought into a position of moderate adduction, which was followed by rapid short to-and-fro excursions.

On passing within the confines of Spencer's area for arrest of respiration, it was found that in the peripheral parts of this area there exist three foci, excitation of which affects the cords in different ways. The most anterior of these foci is responsible for arrest of the cords in adduction; that is, in the expiratory stage of their excursion. Excitation of the focus behind this, corresponding probably to Horsley and Semon's abductor centre in the cat, is followed by arrest of the cords in abduction, that is, in their inspiratory position; whilst stimulation of the most posterior focus, which is situated about the junction of the anterior composite and anterior Sylvian convolutions, results in intensification with acceleration of the movements of the cords. Excitation of Mr. Spencer's chief focus for arrest of respiration on the olfactory lobe resulted in arrest of the cords in the position they occupy during expiration in dogs, and in the position they occupy during inspiration in cats.

In no instance in the whole of the experiments of Semon and Horsley, and Risien Russell, was there any indication of unilateral representation of the cords; on the contrary, excitation of the centre on either side produced an equal abduction effect on both cords alike. The experimental evidence on this point was corroborated by a remarkable case of Jacksonian epilepsy observed by one of us (W. W.), in which the patient, after a fit, while remaining perfectly intelligent, was the subject of complete motor aphasia, being unable to utter a single word, although he could produce inarticulate sounds; in him adduction and abduction of the vocal cords were found to be perfectly normal and bilaterally equal.

Another point of interest has been investigated by Dr. Risien Russell, namely, the inhibition of antagonistic muscles by electrical excitation of the cerebral cortex, on the lines adopted by Professor Sherrington with regard to antagonistic muscles in other parts of the body. This was tested by first dividing the fibres in both recurrent laryngeal nerves, leaving the abductor fibres intact, and then exciting the adductor centre with strong induced currents; but no evidence of inhibition of the abductor muscles was obtained.

Before entering on the discussion of the various forms of laryngeal motor disturbances, it will be well to refer to the law established by one of us (F. S.), namely, that in all progressive organic lesions of the centres or trunks of the motor laryngeal nerves the abductors of the vocal cords succumb much earlier than the adductors. Although a large number of such cases of progressive organic disease acting upon the whole of the nerve-trunk have been recorded, and publicly shewn, in which the abductor muscles had undergone degeneration and atrophy, either alone or at any rate more advanced than in the adductors, one specimen only has yet been demonstrated which, under similar conditions, exhibited the opposite order of events in the development of degenerative changes in the individual laryngeal muscles: this is the case described by Prof. Saundby, in which an extensive carcinoma of the oesophagus invaded both recurrent laryngeal nerves. The adductors succumbed *before* the abductors. During life it was seen that adduction of the cords gradually became more and more impeded, whilst the autopsy shewed that the posterior crico-arytaenoids were less degenerated than any of the other laryngeal muscles supplied by the recurrent nerves. Whether—which is most probable—an anomaly of innervation existed in this case, or what else was the cause of this curious exception, was not cleared up. At any rate there cannot be any doubt that the order of events in this case was the reverse of the ordinary one.

To explain this difference between the abductor and adductor muscles various hypotheses have been advanced. Thus, Sir W. R. Gowers considered it might be a consequence of the advantage at which the most important adductor—the lateral crico-arytaenoid muscle—works in comparison with the abductor (in so far as the former goes in at a right angle, the latter at a very acute angle towards the muscular process of the arytaenoid cartilage), which renders the adductors capable of a longer resistance to disabling influences affecting the whole nerve-trunk. Grützner appears inclined to regard the adductors as belonging to the class of "white," and the abductors to the class of "red" muscles; and suggests that the difference in the muscles accounts for the difference in susceptibility to degenerative processes. Krause's suggestion, viz. that the pathological process underlying the median position assumed by the vocal cords, under the conditions now referred to, did not consist in a primary paralysis of the abductor muscles followed by a paralytic contracture of the antagonists, but in a primary neuropathic contracture of all the muscles supplied by the recurrent, with preponderance of the adductors, has been in recent years abandoned by the author himself, and Wagner's and Grossmann's attempts to trace the position of the affected vocal cord in the middle line to tonic contraction of the crico-thyroid muscle have never met with general acceptance. On the other hand, a number of facts go to shew that in all probability we shall have to look to physiological chemistry for the solution of the mystery. Thus, from Semon and Horsley's experiments on different species of animals, it appears that (*a*) the abductors are the first of all the laryngeal muscles proper to lose their excitability after death; and (*b*) that, when an animal is killed a week after thrusting a thread saturated with chromic acid solution through a recurrent nerve, the corresponding posterior crico-arytaenoid muscle is the first to lose its excitability. Again, Dr. Risien Russell has shewn (*α*) that the abductor and adductor fibres in the recurrent laryngeal nerve are collected into several bundles, the one distinct from the other, and each preserving an independent course throughout the nerve-trunk to its termination in the muscle or muscles which it supplies with motor innervation; (*β*) that when the abductor and adductor fibres are exposed in the living animal to the drying influence of air in exactly similar circumstances, the abductor fibres lose their power of conducting electrical impulses very much more rapidly than the adductors; in other words, that they are more prone to succumb than are the adductors. Hooper's observation, corroborated and explained by Horsley and Semon, that ether has a peripheral and differential effect upon the laryngeal muscles which can be produced only by means of the circulation, the fact that the abductor muscles die sooner than the adductors, and the fact, demonstrated by B. Fränkel and Gad, that gradual cooling of the recurrent laryngeal nerve paralyses the crico-arytaenoideus posticus sooner than the glottis-closers—all these facts, taken together with the clinical and pathologico-anatomical experiences concerning the earlier destruction of the abductors in progressive organic lesions, imply that there is a positive difference in the biological composition of the laryngeal muscles and nerve-endings; whilst the fact that in central (bulbar) organic affections also, such as tabes, the cell groups of the abductors succumb earlier than those of the adductors, points to the probability that there are similar differentiations in the nerve-nuclei themselves. The phenomenon, hitherto obscure, will very likely be explained by biological differences between the components of the laryngeal nerves and muscles. This would constitute an addition to our knowledge of nervous morphology. We formerly knew that differences existed as regards the irritability and power of resistance of the sensory and of the motor nerves, but we assumed complete equality among motor nerves. It is now clear that differences of a more subtle kind exist among these nerves and the physiological conditions of the muscles they supply.

Since the first edition of this work was written there have been numerous further contributions towards the elucidation of the curious law just discussed. To name a few only: Kuttner, Katzenstein, Grabower, Grossmann, Broeckaert, have worked at it experimentally. But whilst incidentally some interesting physiological details have resulted from their labours, it cannot be said that the question of the greater vulnerability of the abductor fibres in the face of organic disease has been brought any nearer its solution than has been stated in the last paragraph; and, indeed, whilst gratefully registering the labours of the authors just mentioned, and of others who have studied additional problems connected with the innervation of the larynx, such as Onodi, Réthi, Massei, Schultz and Dorendorff, Jörgen Möller and Fischer, van Gehuchten and Bochanek, van Bieroliet, de Beule, we may amplify this statement to the effect, that according to our honest conviction the physiological investigations of the last ten years concerning the innervation of the larynx have—however meritorious in themselves—hardly advanced our clinical understanding of the pathology of the laryngeal nerves.

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Spasmodic Affections.—Laryngeal spasms may be due to affections of the nerve-centres, nerve-trunks, or single nerve-twigs. With regard to the nerve-trunk affections, considering that stimulation of the peripheral end of the cut recurrent laryngeal nerve (that is, of all its fibres) results in adduction of the corresponding vocal cord, it is quite possible that, in cases of so-called spasm of the glottis of peripheral origin, not the adductors only, but also the abductors may be in a state of spasmodic contraction; the former, however, preponderating.

The various spasmodic affections may be conveniently divided into two groups:—(i.) Respiratory glottic spasm, and (ii.) Neuroses of coordination.

Respiratory glottic spasm.—**Laryngismus stridulus.**—*Etiology.*—This affection is almost invariably associated with rickets, and occurs chiefly in children from six months to two years of age or up to the eighth or ninth year.

Whilst the remarkable excitability of the nerve-centres in rickets disposes to the affection, the spasms are often excited directly by some reflex irritation in the alimentary tract, such as undigested food or parasites; or it may be due to post-nasal adenoids, or to sources of excitation such as teething, a pendulous epiglottis, large bronchial glands, or possibly an enlarged thymus. It is sometimes directly brought on by emotion; and it is very likely that defective nutrition and consequent irritability of the cortical adductor centres may cause laryngismus (Semon and Horsley). This would also explain the "carpo-pedal" contractions, general convulsions, and so forth, which not rarely accompany laryngeal spasm in children, and which the authors just named consider as an overflow of energy from the irritated laryngeal adductor centre or centres to the neighbouring centres. The patients usually are ill-nourished, unhealthy children.

Laryngismus may arise as a complication of measles or whooping-cough, especially in children otherwise predisposed; and whooping-cough in particular leaves a strong disposition to laryngeal spasm for some months after its own disappearance.

Symptoms.—In a well-marked attack, after a few stridulous inspirations, short at first but gradually more prolonged, spasmodic closure of the glottis occurs, the respiratory movements of the chest and respiration ceasing absolutely. The child presents a most painful aspect, with the head thrown back, the neck forward, the eyes staring, the pupils contracted, and the countenance bearing an expression of extreme anxiety, at first flushed, then in a few seconds pallid or livid; the veins of the neck are swollen, and perspiration gathers on the face. The glottic spasm lasts from fifteen seconds to two minutes, and the glottis may remain closed till loss of consciousness or even death occurs. The attack, if not fatal, ends as it began with a few short stridulous inspirations, either continuous or intermittent, as in sobbing. In severe cases these symptoms are accompanied by spasms of the facial muscles, and by spastic, so-called carpopedal contractions; in these the thumbs are turned in and flexed on the palms and the fingers closed over them or rigidly extended; the carpal joints are turned inwards, the feet somewhat flexed and turned inwards. In some cases general convulsions supervene on these phenomena. In the less severe forms, the carpopedal spasms are absent and the symptoms less pronounced, the parents often speaking of the attacks as "passion-fits" or "holding the breath." Generally as soon as the attack is over the child resumes its play, and seems as well as ever. These attacks may occur very occasionally, or they may follow one another in quick succession; generally there are one or two attacks daily.

Prognosis.—In very severe cases death from asphyxia is by no means rare, and the prognosis should therefore be guarded, although the mean mortality of all cases is very small. From a therapeutic standpoint the prognosis is generally distinctly favourable, especially when there is a prospect of removing the underlying cause, as in rickety children who constitute the vast majority of the cases; yet some cases are very persistent, and, particularly in those which result from pressure by an enlarged bronchial gland, the attacks are liable to recur till the child has attained the age of eight or nine, or even more. In the "silent cases"—those in which there is no inspiratory stridor—the prognosis is especially grave.

The *diagnosis* of laryngismus stridulus rests upon the suddenness of the attack, the complete cessation of the respiratory movements at the height of the attack, the absolutely free intervals, and the absence of symptoms of inflammatory disease in the larynx, such as cough, hoarseness or aphonia, fever, and so forth.

Spasm of the glottis in adults is generally a reflex phenomenon brought about by irritation of a vagus or, in very rare instances, of both recurrent laryngeal nerve-trunks by aneurysms or mediastinal growths and the like, or by direct irritation of the larynx by foreign bodies, neoplasms, adenoid hypertrophy of the lingual tonsil, an elongated uvula, and so on. Glottic spasm also occurs in certain lesions of the nerve-centres, as in the laryngeal crises of locomotor ataxia, in hydrophobia (in which, according to a very interesting observation made by Dr. Newton Pitt, the abductors only of the vocal cords appear to be affected by the spasm), tetany, and hysteria.

The *symptoms* are usually much less severe, though of the same character as in infants and children; they often amount to no more than a succession of stridulous inspirations.

In other cases, however, the spasm may be prolonged till consciousness is lost, or even life itself suspended. In very rare cases, according to some authors, there is a slight but constant spasm. In hysteria it may occur either in the paroxysmal or in the more continuous form: in the latter, which has also been termed functional inspiratory spasm, the vocal cords, instead of separating on intended inspiration, approach each other, remain together during the inspirations so that the air enters with difficulty and stridor through the narrowed glottis, and only separate to some extent during expiration.

Treatment.—As a rule, the spasm passes off spontaneously after a few seconds; but prompt measures should be taken to shorten the attack as far as possible by removing any tight garments, opening the window, placing the patient in the semi-recumbent position, and applying cold water to the face and head and smelling-salts to the nostrils, while the legs and body may be immersed in a hot bath. If asphyxia be threatening, tracheotomy should be performed without delay, followed, if necessary, by artificial respiration.

The general treatment depends on the exciting cause of the neurosis. Warm clothing, fresh air, simple diet, and avoidance of mental excitement or hard brain-work are of first importance. Faecal accumulations, or intestinal parasites, when present, must of course be removed. Above all, if any indications of rickets are noted, treatment must be directed to overcome this condition by the administration of cod-liver oil, and especially of small doses of phosphorus. In strumous children the syrup of the iodide of iron and cod-liver oil will be useful. If the attacks recur frequently, bromide of potassium, continued for some length of time, belladonna, or chloral will tend to keep them off and render them less severe. In a case observed by one of us (F. S.) the use twice daily of a 2 per cent spray of cocaine, directed to the larynx, succeeded—probably by gradually diminishing the peripheral hyper-irritability—in causing complete disappearance of attacks of very serious laryngeal spasm in a gouty adult within a fortnight.

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Neuroses of Co-ordination.—(*a*) *Choreic movements* of the vocal cords may accompany general chorea; and have also been noticed independently. We here refer to disorderly action of the cords in contradistinction to the glottic spasm with forced expiration in cases of "barking cough." In disseminated cerebrospinal sclerosis a tremulous action of one or both cords, similar to the tremors of the limbs on intended movements, is sometimes present.

Functional inspiratory spasm has already been referred to as one of the forms of hysterical laryngeal spasm, the vocal cords coming together on inspiration, and separating but slightly on expiration. The symptoms in these cases are very similar to those of bilateral paralysis of the abductors; but when the vocal cords are watched by the laryngoscope during expiration they are occasionally seen to separate well. This affection appears to occur only in nervous or hysterical persons, though a minor degree of it is often witnessed in nervous people examined with the laryngoscope for the first time; the vocal cords in such cases are approximated instead of separated on attempted inspiration. Very occasionally this form is associated with spastic aphonia (see *c*). Psychical treatment, bromide of potassium, the cold douche or intralaryngeal faradic current usually effect a cure.

(*b*) *Nervous Laryngeal Cough.*—There is a condition in which spasmodic closure of the glottis appears in the form of separate, sudden, short contractions of the adductors, in association with similar contractions of other respiratory muscles, which results in an extremely loud, harsh, abrupt cough, the "barking cough of puberty" (Sir Andrew Clark). It occurs in young persons of both sexes. We have seen more men than women affected by it, and it is not limited to the period of puberty; it is most common between sixteen and twenty, but the ages of the patients vary from ten to twenty or more. The cough generally ceases during sleep, though not always; usually it is single, not a series of successive coughs, in which character it differs from the cough due to sensory laryngeal irritation; throughout the day it recurs persistently, even during rest. This nervous laryngeal cough is not associated with any demonstrable lesion, and the voice is not in any way impaired; there is no shortness of breath involving forcible inspiration after the cough. In fact, it is simply a sudden closure of the glottis, with a forcible expiration, due to affection both of the laryngeal and pulmonary branches of the vagus. The general health is curiously little affected, and the cough often appears to be a much greater nuisance to the patient's family than to the sufferer himself.

This affection is really one of the "convulsive tics"; and is not in any way associated with volitional acts. It may last for weeks, months, or even years, but finally almost always ceases spontaneously.

Nervous laryngeal cough is very little amenable to ordinary treatment. The remedy which, with one single exception, has best answered in all the cases observed by one of us (F. S.) is a sea-voyage, which usually acts like a charm within a few days. Removal of the patient from home, a stay at the seaside, general sedatives, and the like, are not to be compared in efficiency to a sea-voyage, which should be urged upon the patient's friends, however great the difficulties. If a sea-voyage be quite impossible, the internal use of bromides in large doses [sulphate of iron—Ed.] and local cocaine applications may be tried.

(*c*) *Phonic Spasm* (Dysphonia spastica).—This is a form of contraction of the adductors, originally described by Schnitzler, which is probably always allied to a similar contraction of the tensors of the vocal cords and of the thoracic expiratory apparatus, and only occurs on attempted phonation. The affection is analogous to writer's cramp, and one of us (F. S.) has seen a case of spastic aphonia associated with similar spasm of the masseter and orbicularis oris; another coexisted with writer's cramp. This form of glottic spasm, like the preceding, from which it differs in that it only appears on attempted phonation, is rare. It is a disease of adult life and almost always occurs in highly-strung men who have to use their vocal organs professionally (especially clergymen), so that it may be classed amongst the professional neuroses. Occasionally, however, both men and women in robust health, whose occupation is of a silent kind, may be attacked.

In its earlier manifestations the patient, after producing a few words, especially when using the voice in a professional capacity, such as preaching or reading the lessons in church, suffers from notable impairment or complete loss of voice. As the disease increases, any attempt at phonation results in spasmodic closure of the glottis, and the words are lost in fruitless attempts to force a current of air through the closed part. The voice in these circumstances assumes such a curiously oppressed character that even one who has never before seen a well-marked case of the disease may be enabled to diagnose subsequent cases from the particular timbre of the voice alone. Laryngoscopically the vocal cords are seen to act normally during respiration; but on attempted phonation the cords come into complete apposition; in fact, so forcibly are they adducted that they may seem to overlap one another, and one arytaenoid cartilage may push itself in front of its fellow. The spasm lasts as long as attempts are made to speak; but as soon as voluntary effort at phonation ceases the glottis opens. Whispering is sometimes less difficult and may be possible. Respiration is free and noiseless.

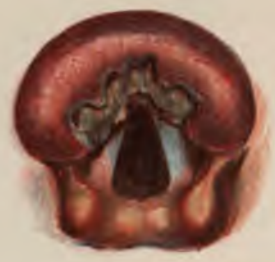
To this class belong the cases described by B. Fränkel under the name *mogiphonia*, in which spasm with impairment or loss of voice occurs only on singing or attempts at public speaking, the ordinary conversational voice being unimpaired.

Treatment in these cases, in our experience, is almost always futile. All the remedies usually recommended in textbooks fail—tonics, electricity, rest of voice, hydropathic treatment, sea-voyages, and so forth. The only method from which any improvement may be hoped for, and this in the earliest stages only, consists in rational breathing and elocutionary exercises: it is characteristic of these patients that they almost always attempt to pronounce or to read long sentences without taking an intermediate inspiration. Quite recently one of us (F. S.) has seen two patients actually cured after a persevering course of breathing and speaking exercises, patiently carried out by a scientific-minded teacher of elocution; in another recent case, however, this method failed too.

(*d*) *Laryngeal Vertigo.*—There is a curious and rare form of spasm of the larynx, followed immediately by vertigo and loss of consciousness, to which Charcot originally applied this name. He considered it to be analogous to Menière's disease, the afferent nerve being, according to his view, the superior laryngeal. The views of its pathology differ widely; thus, Krishaber regarded the vertigo as due to spasm of the glottis and arrested action of the respiratory muscles, and Dr. Gray looks upon the affection as a form of epilepsy. Dr. M'Bride explains the phenomena by the action of forced expiration with a closed glottis; he made experiments on the effect of forced expiration under these conditions, and found that the sphygmographic tracings of the pulse shewed a rapid and continuous diminution of the up-stroke. Whilst it may be pointed out that the effect on the pulse is probably due to increased intrathoracic pressure on the pulmonary vessels, Dr. M'Bride argues that in laryngeal vertigo there is complete closure of the glottis, and thus the whole expiratory effort is felt, through the air contained in the lungs, by the alveoli, the large blood-vessels in the thoracic cavity, and the heart itself. As a result syncope—or a tendency to syncope—is produced, but almost at the same moment the spasm of the glottis relaxes and the attack is over.

In a case observed by one of us (W. W.) the patient, a boy, seemed to get an attack whenever pressure was made on the laryngo-tracheal region,

PLATE XI



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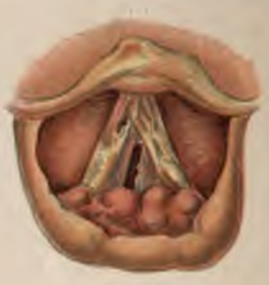
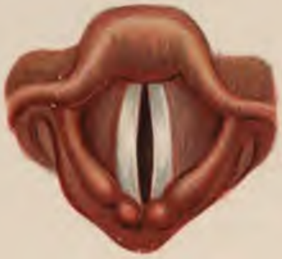


Fig. 2.



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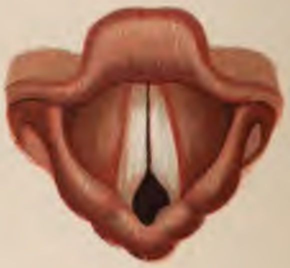

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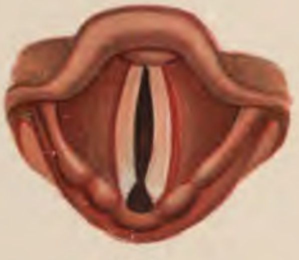

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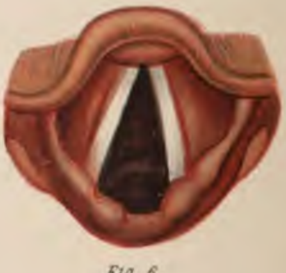

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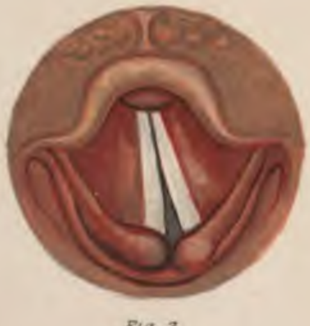

Fig. 7.

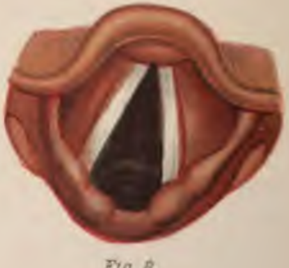

Fig. 8.

PLATE XI.

SYPHILIS OF THE LARYNX, AND LARYNGEAL PARALYSIS.

Fig. 1. Tertiary syphilis of the larynx. A gumma of the epiglottis is breaking down—shewing deep crateriform ulceration.

,, 2. Tertiary syphilis. The vocal cords have been the seat of former ulceration, which has now cicatrised into partial union of the cords. The posterior boundary of the glottis is occupied by pinkish-yellow syphilitic mammillated outgrowths.

,, 3. Paralysis of the thyro-arytaenoidei interni.

,, 4. ,, ,, inter-arytaenoideus.

,, 5. Combined paralysis of the thyro-arytaenoidei interni and inter-arytaenoideus.

,, 6. Complete recurrent paralysis of the left vocal cord—during inspiration.

,, 7. Complete recurrent paralysis of the left vocal cord—during phonation. The right vocal cord crosses over to meet the paralysed left cord.

,, 8. Abductor or posticus paralysis of the left vocal cord—during inspiration.

as for instance, when another boy dragged on his collar from behind. The attacks began with a few short spasmodic coughs, coming especially on attempted phonation. There was no inspiratory stridor. He became unconscious after a few moments; at first pale, he soon got cyanosed. The thumbs were adducted, but there were no convulsions or general twitchings. He generally passed much urine after the attacks. He had been very subject to "croup" in early childhood. Possibly these attacks were epileptic in nature.

The patient may seem to be in perfect health; or he may suffer from a catarrhal affection of the nasal passages. A tickling sensation in the larynx results in an effort to cough. This is immediately followed by giddiness and obscurity of vision, and the sufferer falls down in a state of unconsciousness lasting a few seconds only, from which, as already stated, recovery is immediate and complete. The face is either pale or turgid—there may be slight twitchings of the face or limbs. In slighter cases consciousness may not be completely lost, the seizure terminating only with the occurrence of the vertigo. The attacks recur at intervals varying from a few days to months.

As regards treatment, the main indications are to improve the general health by rest and change of air, and by the administration of cod-liver oil and general nervine tonics such as iron, quinine, phosphorus, and arsenic; the tendency to spasm may be averted by bromide of potassium and similar remedies. Any catarrhal affection of the respiratory tract should be removed by appropriate treatment.

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PARALYSIS.—Motor laryngeal paralysis may be due to—

(1) Degenerative changes in the nuclei of the laryngeal motor nerve-fibres in the floor of the fourth ventricle;

(2) Degeneration, injury of, or pressure on the vagus trunk, or its superior and recurrent branches;

(3) Functional neuropathic impairment;

(4) Paralysis which, in its initial stages at any rate, may be reflex;

PLATE XI

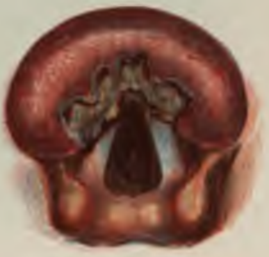

Fig. 1.

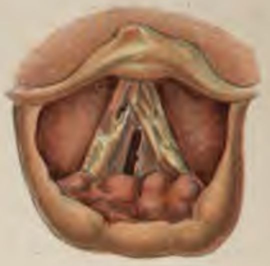

Fig. 2.

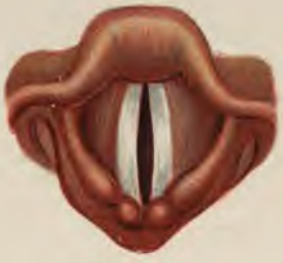

Fig. 3.

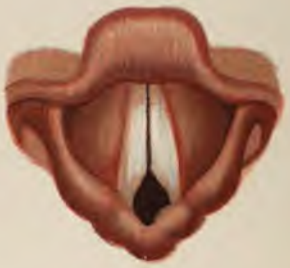

Fig. 4.

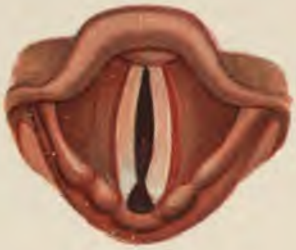

Fig. 5.

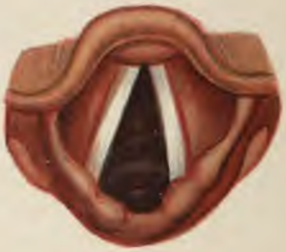

Fig. 6.

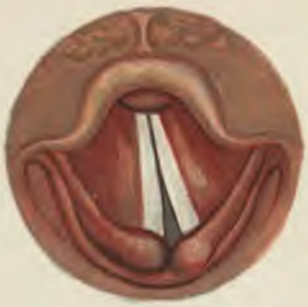

Fig. 7.

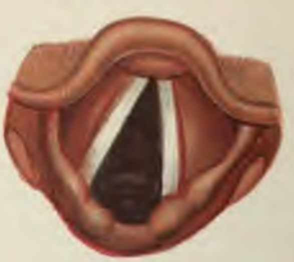

Fig. 8.

the result may be bilateral paralysis of the abductors of the vocal cords; a reflex paralysis ingeniously explained by Sir George Johnson as the result of a centripetal irritation of the trunk of the vagus acting on the nervous centre, and through it upon the nerve-supply to the laryngeal muscles of the opposite side.

The left recurrent nerve is more frequently affected than the right; and the most frequent cause of this paralysis is aneurysm of the arch of the aorta; the right recurrent is more liable to compression by pleuritic thickening accompanying tuberculosis of the right lung and by aneurysms of the innominate, though it also may suffer from aortic aneurysm. The left recurrent nerve branches off from the left vagus on a level with the concavity of the aortic arch, and winds round it from before backwards to ascend to the larynx; whilst the right recurrent begins on a level with the right subclavian artery, around which it winds before passing upwards. Thus not only is the left recurrent very liable to be affected in the earlier stages of aneurysms of the aortic arch, even before there are any other manifestations of aneurysm, but as both the recurrent and the vagus above its recurrent branch have the longer course within the chest on the left side, there is also greater liability for the left cord to be affected by other intrathoracic tumours.

If any of these causes act on the recurrent nerve of one side, unilateral abductor paralysis results at first; whereas if the conditions obtain on both sides—that is, if there be a bilateral incomplete lesion of the bulbar centres, or of the trunks of both recurrent laryngeal nerves, or if there be pressure on one pneumogastric only with resulting reflex paralysis—bilateral abductor paralysis will result. Of course if the paresis result from interference with the vagus trunk above the superior laryngeal branch, anaesthesia of the larynx will be present, in addition to motor paresis. If the lesion be high up and due to a tumour, or to diffuse pachymeningitis, other cranial nerves, such as the spinal accessory, glosso-pharyngeal, and hypoglossal, may be involved.

In unilateral abductor paralysis the affected cord remains fixed in the median line, that is, in the position of phonation (Fig. 8, Plate XI.); and as the opposite cord is unaffected, respiration is not embarrassed unless the cause of the paralysis simultaneously produces direct compression of the lower air-passages, as it may in cases of goitre and of aortic aneurysm. In such circumstances, that is to say, in the initial stages of all the severe lesions mentioned as "causes" which may implicate the laryngeal nerves—and indeed not rarely up to the patient's death—neither vocal nor respiratory symptoms need occur in adults: thus the laryngeal lesion, which may be of the greatest importance for the correct diagnosis of the whole case, will entirely escape notice unless it be a part of routine practice to examine all cases in which lesions of the laryngeal nerves could occur, whether there be symptoms pointing to the larynx or not.

In bilateral abductor paralysis both cords are defective in abduction on inspiration; and when the abductor paralysis is complete, the cords remain in or near the median position by the gradual supervention of paralytic contracture of the adductors, a very small chink only being seen between them. Laryngoscopically this looks like a continuous position of phonation. Inspiration is, of course, greatly embarrassed in complete paralysis; but fortunately bilateral abductor paralysis is often only partial; or while one cord is affected by complete abductor palsy the other is only partially palsied. Paroxysmal attacks of urgent dyspnoea, with characteristic stridulous inspiration, are prone to occur on slight exertion or mental excitement, and may at any time end in sudden and fatal asphyxia. In the intervals there is sonorous or stridulous inspiration, particularly in sleep; but expiration is free and the voice normal.

The prognosis of bilateral abductor paralysis is obviously very grave, and at any moment tracheotomy may be necessary. In progressive lesions the adductors may eventually become involved; and with the complete paralysis of the cords, which then assume the cadaveric position, respiration becomes less impeded, whilst the voice becomes impaired and finally quite aphonic. Such secondary implication of the adductors may not occur for several years; and, as the voice meanwhile is in no way impaired, slighter degrees of bilateral abductor paralysis may exist without the slightest suspicion of such a disorder on the part of the patient, particularly in cases in which the patient is unable to make strong muscular efforts, as in the more advanced stages of tabes dorsalis.

(iii.) *Complete recurrent paralysis* (that is, involving all the muscles supplied by the recurrent laryngeal nerves) of the vocal cord results from lesions which are equivalent to a transverse section of the nerve affected. Any of the lesions mentioned as causes of abductor paralysis may give rise to complete recurrent paralysis. Probably abductor paralysis is almost always present for a longer or shorter time in the earlier stages of pressure on a recurrent nerve; but sooner or later the adductors are also involved.

If only one nerve is paralysed, the respiration is not affected, and the voice is either aphonic, hoarse, or sometimes almost normal when the healthy cord "compensates"—that is, crosses the median line in phonation to join its paralysed fellow (Fig. 7, Plate XI.); but it is apt, in such circumstances, to break into falsetto. During quiet respiration the larynx appears nearly normal, but in phonation the healthy cord is sometimes over-adducted and passes across the middle line to meet the paralysed cord, producing a peculiar distortion of the laryngeal image, the position of the glottis being oblique. The arytaenoid cartilage on the paralysed side being unsupported by its muscles, may be pushed aside so that it lies behind the sound and over-adducted arytaenoid. In deep inspiration the paralysed cord and its arytaenoid remain immobile in the cadaveric position, whilst the arytaenoid on the healthy side passes farther back.

In bilateral complete recurrent paralysis, which is extremely rare, the vocal cords remain in the cadaveric position both during phonation and inspiration. There is no dyspnoea during rest, but there is complete aphonia. It is usually the result of pressure on both recurrent nerves; or represents the final stage of laryngeal paralysis due to central nerve lesions, as in tabes, syphilitic nuclear disease, and the like; but it may be due to any of the causes enumerated under the head of abductor paralysis. It is seen most frequently—comparatively speaking—in cases of carcinoma occupying the plate of the cricoid cartilage and the adjoining lower regions of the front wall of the oesophagus.

(iv.) *Paralysis of the thyro-arytaenoidei interni*, or internal tensors of the vocal cords, is usually bilateral; and is most frequently the result of over-straining the voice; or of catarrhal laryngitis, especially in anaemic and neurotic persons. The vocal cords are practically the tendons of the thyro-arytaenoidei interni muscles which are inserted into their whole length. The function of these muscles is to render tense the free margin of the vocal bands; when therefore they are weakened, or paralysed, the vocal cords lose their normal flat appearance and become rounded and narrowed; thus they cannot approximate perfectly, and a narrow elliptical space, extending throughout the length of the glottis, is left between the cords during phonation, which consequently is weak and husky; or the voice may even be lost (Fig. 3, Plate XI.). The thyro-arytaenoidei muscles are often paralysed in central nerve-lesions, and their paralysis is often associated with or follows next (F. S.) upon paralysis of the glottis-openers. It is, however, important to remember that some elliptic gaping of the vocal cords during phonation is by no means rarely seen in persons who are in full possession of their voice.

(v.) The *inter-arytaenoideus* muscle may be paralysed alone in catarrhal conditions and in hysteria. The paralysis is always bilateral, and the voice is generally much impaired, or even quite lost. In these cases the anterior three-fourths of the vocal cords are seen to come together on attempted phonation, while a triangular chink is left between the vocal processes (Fig. 4, Plate XI.). Paralysis of the thyro-arytaenoidei muscles may be associated with paralysis of the inter-arytaenoideus, giving a characteristic double elliptic glottic chink (Fig. 5, Plate XI.).

B. **Paralysis of the Muscles supplied by the Superior Laryngeal Nerve.**—*Isolated Paralysis of the Crico-thyroid Muscles.*—The function of the crico-thyroid muscles is to render the vocal cords tense on phonation; they are the external tensors of the vocal cords. Paralysis of the crico-thyroid alone is very rare. According to Mackenzie, it may be caused by cold or over-strain of the voice; and it is characterised by a wavy outline of the glottis with a slight depression of the central portion of the vocal cords in inspiration, and a corresponding elevation in expiration and vocalisation (see also Sensory Laryngeal Neuroses, p. 278). In unilateral paralysis of the crico-thyroid muscle the corresponding vocal cord stands on a lower level than its fellow. If the whole of the superior laryngeal nerve be paralysed, the motor impairment will be accompanied by anaesthesia of the corresponding side of the larynx. Mackenzie also pointed out that crico-thyroid paralysis can be detected by applying the finger to the crico-thyroid space on either side during phonation, when a want of tension will be felt.

For the treatment of paralysis of the superior laryngeal nerve and the muscles it supplies see Sensory Neuroses (p. 278).

Diagnosis.—The significance of laryngeal paralysis is of very considerable importance in medical practice, not only on account of the symptoms that may be produced, or the danger to life that may be involved in various forms of paralysis, but still more on account of the valuable aid to the diagnosis of many obscure intrathoracic or central nerve affections that may be afforded by a due appreciation of the pathological source of the laryngeal condition.

Even when the impaired movements of the vocal cords are undoubtedly due to local causes, it is necessary to distinguish between true neuroses, myopathic palsies, and the simulation of palsy by fixation or impaired freedom of action in the crico-arytaenoid joint; or the impairment may be the earliest indication of early malignant disease in the cords or in their immediate neighbourhood. Local paralysis due to neuritis is generally of diphtheritic or rheumatic origin; myopathic paralyses mostly follow catarrhal inflammations. Mechanical fixation of the arytaenoids by cicatricial contraction of the mucous membrane, after ulcerative diseases or injuries, may account for the immobility of the cords. Any thickening in the neighbourhood of the arytaenoid cartilage, or abnormal distensions of the folds of mucous membrane, or tumefaction at the base of an immobile arytaenoid cartilage, are in favour of mechanical fixation. In unilateral recurrent paralysis the arytaenoid cartilage on the paralysed side may be displaced by the sound and over-adducted arytaenoid cartilage.

Bilateral ankylosis is rather liable to give rise to error in diagnosis, inasmuch as it may so closely resemble bilateral recurrent paralysis; but complete paralysis of both cords (apart from ankylosis) is extremely rare.

Abductor or complete paralysis, whether unilateral or bilateral, may be the earliest symptom of a thoracic tumour—and especially of an intrathoracic aneurysm, of malignant disease of the oesophagus, intracranial disease, tabes, disseminated cerebrospinal sclerosis, or general paralysis; even although all other signs be still absent. The possibility of any of these conditions being the cause of the paralysis should ever be present in the mind of the physician, who will endeavour to detect further indications of their existence. Points in favour of bulbar lesions are—(*a*) Persistent increased pulse frequency without any pulmonary affection or febrile disturbance to account for it; (*b*) Implication of both cords: but it is particularly to be noted that the unilateral character of the paralysis does not in itself indicate that the disease is peripheral; (*c*) Coexisting paralysis of the soft palate and tongue.

The **treatment** of laryngeal paralysis will depend upon the nature of the chief cause of the laryngeal condition. When it is due to pressure on a nerve-trunk or to central nerve disease, the prognosis is generally most unfavourable. In any form of organic laryngeal paralysis the chief indication is, if possible, to remove the cause of the mischief. When it is caused by syphilitic disease the usual antisyphilitic treatment is indicated; if enlarged glands or tumours are pressing on the nerve-trunks it may be possible to remove them (this applies particularly to goitre); but when the pressure is within the thoracic cavity, we can rarely hope to cure the paralysis by operative or medicinal treatment. In advanced bilateral abductor paralysis, since at any moment sudden and fatal asphyxia may arise, tracheotomy ought always to be proposed, not as a curative but as a prophylactic measure, pending the adoption of any further treatment by which we may hope to obtain a permanent cure, in which event the tube can be removed. A cure may be possible when the paralysis is due to pressure, as in goitre, or to syphilis or diphtheria; but not when it is due to bulbar disease, as in tabes or labio-glosso-laryngeal paralysis; though in these bulbar cases complete recurrent paralysis may eventually supervene and render the tracheotomy tube unnecessary. In all cases in which the bilateral abductor paralysis is brought about by pressure within the thoracic cavity, the possibility of a second stenosis lower down, due to pressure on the trachea by the same tumour or aneurysm which is pressing on the nerve-trunk and causing the abductor paralysis, should be borne in mind; and in order to prevent disappointment this should be explained to the patient. A second seat of stenosis is probably present when there is marked expiratory as well as inspiratory stridor, and difficulty in respiration. But when the narrowing of the glottic chink is in itself sufficient to account for the dyspnoea, tracheotomy should be performed; and the low operation should always, if possible, be so chosen that the tube may be inserted below the compressing tumour, if it be in the neck; if it be in the thoracic cavity, it may be possible to pass a long flexible tube down the trachea and past the stenosis.

If the condition is due to maladies amenable to remedies—such as syphilis, or the neuritis of diphtheria or of cold—general treatment must not be neglected, while direct treatment of the paralysis itself, by local faradisation and hypodermic injections of strychnine (gr. $\frac{1}{30}$ gradually increased to gr. $\frac{1}{10}$), should be steadily pursued, in the hope that the conductivity of the nerve may not be wholly lost. In cases of functional paralysis of the adductors, due to excessive use of the voice in anaemic, overworked, or weakly persons, rest and tonic treatment must be enjoined. The patient must abstain from using the voice, live as regularly as possible, avoid all fatigue and mental worry, and take plenty of sleep, food, and open-air exercise, and a sufficiency of cold baths. Iron, strychnine, phosphorus, quinine, and arsenic and similar tonic remedies may be advantageously administered; and locally applications of mild galvanic or faradic electric currents must be applied to the region of the pneumogastrics. In cases due to inflammation, the usual remedies suited to laryngitis may be employed, as well as local faradisation.

In hysterical paralysis of the adductors, emotional effects, or anything that gives a shock to the system, will often produce a cure; and a similar result often follows stimulation of the larynx, as by inhalation of ammonia, the application of a laryngeal brush, and so forth. Nothing is so satisfactory, however, as local faradisation; and, though this may be given by applying an electrode to either side of the larynx externally, it is much more effectual if Mackenzie's endo-laryngeal electrode is used. The current, though not so powerful as to be actually painful, should be fairly strong at the outset; by timid handling the beneficial effects of the shock are often spoiled. One strong application is generally sufficient, but sometimes it has to be repeated once or twice.

In reflex paralysis the eccentric cause, such as uterine disorder for example, should be sought for and remedied.

Paralyses of the arytaenoideus and crico-thyroid muscles, when due to cold or diphtheria, are often very obstinate; and local faradisation at frequent intervals may have to be continued over a long period.

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See also the references under Introductory Remarks; and particularly the chapter on "die Nervenkrankheiten der Kehlkopfs" in Heymann's *Handbuch*, in which a very full bibliography is given up to 1897.

(II.) SENSORY NEUROSES.—**Anaesthesia.**—The superior laryngeal branches of the vagi supply sensation to the mucous membrane of the larynx on each side; and loss of sensibility occurs when these nerves are paralysed. The loss of sensation may vary from slight diminution to complete anaesthesia; and the area affected may be on one side only, or may extend to the epiglottis or supraglottic portions of the larynx; or it may be complete, and invade the whole of the larynx and the upper part of the trachea. The anaesthesia may be due to peripheral lesions, as in diphtheria, syphilis, or injury to the vagus or superior laryngeal nerves; or it may be central in origin, as in bulbar paralysis, locomotor ataxia, general paralysis of the insane, apoplexy, after an epileptic fit, and generally, though in a minor degree, in hysteria.

But the superior laryngeal nerve also supplies motor innervation to the crico-thyroid muscles; and therefore in cases due to peripheral lesions, and sometimes in bulbar and other central nerve lesions, these muscles are paralysed at the same time. Obviously other motor laryngeal paralyses and lesions of other cranial nerves may coexist, according to the situation and extent of the disease.

The *symptoms* consist mainly in a tendency for mucus and food to enter the larynx. The mucous membrane of the larynx itself being insensitive, the particles of food often enter the lower air-passages. When the anaesthesia is complete and subglottic, the larynx does not react by reflex spasm upon the ingestion of food; so that the particles may enter the lower air-passages, and either cause most violent cough—the tracheal mucous membrane having retained its reflex irritability—or obstruct the passage and produce dangerous attacks of suffocation; or, again, they may become impacted in the bronchi and give rise to pneumonia—the "Speisepneumonie" of the Germans. Hence it is also desirable, in all operations in which blood may enter the larynx, not to push the narcosis to the abolition of the cough-reflex.

In one-sided anaesthesia there is a tendency for mucus and saliva to collect on the insensitive side.

The *diagnosis* can only be made with certainty by touching the laryngeal mucous membrane in various parts with a probe under guidance of a laryngoscopic mirror, when defects of sensation and loss of reflex cough are readily detected. It is hardly necessary to emphasise the importance of noting any coexisting paresis or anaesthesia of the fauces, pharynx, or tongue.

The *prognosis* depends on the cause of the anaesthesia; in most cases the prospect of cure will be very remote. Post-diphtheritic anaesthesia tends to disappear spontaneously in the course of five or six weeks; but in all cases, so long as complete anaesthesia lasts, it is a very dangerous affection.

Treatment consists, in the first place, in special care in feeding the patient. In all forms in which anaesthesia is bilateral, food must be given by means of the oesophageal tube only, or by enema. Great care should be observed in introducing the tube, and it should be guided by the forefinger of the left hand lest it enter the open and anaesthetic glottis without producing cough. To be quite certain that the tube is in the correct position, the patient should be told to speak a word or produce a sound before the food is administered, as with the tube in the trachea phonation would be impossible.

Secondly, in those cases which are due to diphtheria, the faradic and galvanic electric current should be applied with one pole to the anterior wall of the pyriform sinus, near which the superior laryngeal nerve runs; and hypodermic injections of strychnine should be given. In syphilitic disease of the central nervous system iodide of potassium and mercurial inunctions are indicated.

Hyperaesthesia, Paraesthesia, and Neuralgia.—Increased sensitiveness of the laryngeal mucosa, tickling and pricking sensations or a sense of a foreign body in the larynx, burning sensations, pressure, pain, constriction, rawness, and other perverted sensations are commonly met with in hysterical, hypochondriacal, or anaemic patients. Sometimes these sensations are set up by a hypertrophied lingual tonsil impinging on the epiglottis, by caseous masses in the tonsillar crypts, or by pharyngitis; for any source of irritation in the pharynx or rhino-pharynx is usually referred subjectively to the larynx: in the majority of the purely neurotic cases, however, the laryngeal symptoms are associated with similar sensations in the pharynx. They are particularly frequent in women at the time of the climacteric period (see Sensory Neuroses of the Pharynx, p. 157).

The result of laryngoscopic examination in these cases is generally negative, or at most reveals an anaemic condition of the mucosa. Hyperaesthesia is often a marked feature in gouty and rheumatic laryngitis; and a similar condition with perverted sensations is sometimes a premonitory symptom of tuberculous disease of the lungs; in all these cases, if there be the slightest suspicion of a tuberculous proclivity, the lungs should be examined by the physician. When associated with central nerve affections, such as tabes, the occurrence of laryngeal crises and the presence of abductor paresis and increased frequency of the pulse should suggest their real nature.

In these affections the indications are to improve the general health by nervine and tonic remedies, sea-bathing, and the like. If the pain be intermittent, and suggestive of neuralgia, quinine or croton-chloral-hydrate may be given, and locally a menthol spray may be used. Treatment, however, in these cases is generally most unsatisfactory, and on no account should the patient become habituated to narcotics. (See also Pharyngeal Sensory Neuroses, p. 158.)

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III.—LARYNGEAL MANIFESTATIONS OF CHRONIC DISEASES OF THE CENTRAL NERVOUS SYSTEM.—In *tabes dorsalis* the medulla oblongata is very often invaded; and among bulbar nerves the vago-accessory is by far the most frequently attacked. Hence the laryngeal nerves are very frequently affected. The various conditions that may arise are:—(i.) Sensory disturbances, such as anaesthesia, hyperaesthesia, paraesthesia, and the various abnormal sensations that precede or accompany laryngeal crises, such as tickling, constriction, inclination to cough, and in some instances anaesthesia. (ii.) Incoördination of the laryngeal muscles or of the vocal cords. The voice may be thick and jerky, or it may suddenly disappear after a few words have been uttered, as in dysphonia spastica. On attempted phonation, as observed by Krause, the cords may be suddenly adducted, then remain for a short interval in the semi-adducted position, and finally become adducted in the median line; during inspiration the cords, after being strongly adducted, are suddenly abducted to an extreme degree. Burger has drawn attention to the analogy between these irregular movements of the vocal cords, on attempted phonation or deep inspiration, and the ataxic movements of the lower extremities, in which the voluntary movements are very irregularly accomplished. (iii.) Laryngeal crises are frequently present in locomotor ataxia, particularly in its earlier stages, and may indeed constitute the earliest manifestation of this disease. In a considerable proportion of cases they are associated with abductor paresis. They tend to become less severe and less frequent as the paretic condition becomes more marked. The onset of an attack is usually preceded by a sense of tickling in the larynx, with tendency to cough, quickly followed by a sense of constriction and dyspnoea due to the spasmodic closure of the glottis. A succession of abrupt coughs, resembling whooping-cough, continues till the patient feels almost asphyxiated; and is followed by inability to inspire, or by a long-drawn whoop, during which air is drawn into the chest with very great difficulty. The whole attack may last but a quarter of a minute, or may persist for five or ten minutes. Death from asphyxia is unusual but is not unknown. In some cases the laryngeal crises are attended by sneezing, vomiting, vertigo, pains in the chest and limbs, or even by general convulsions and loss of consciousness. They may also be associated with gastric crises. (iv.) Paralysis, usually of the abductors of the cords, unilateral and bilateral. The symptoms of abductor paralysis were described on p. 271.

After abductor paralysis has lasted for some time it may be followed by complete recurrent paralysis; but it should be noted that abductor paralysis may be the first, and for a long time the only demonstrable, sign of tabes, and that adductor paralysis may not appear until the abductor paralysis has for many years been associated with the supervention of many definite symptoms of tabes. Thus, one of us (F. S.) has met with a case in which the abductor paralysis had existed twelve years at least, and yet, though paralysis of the internal tensors—the thyro-arytaenoidei—had occurred, the adductors were still unaffected. (The internal tensors, as already mentioned, are the muscles next in order to the abductors to succumb to progressive organic disease.)

In tabes, in association with abductor paresis, the pulse-rate is very often persistently accelerated. This is due to the fact that the inhibitory nerve of the heart, like the motor nerves of the larynx, is derived from the accessory nucleus.

No necessary connexion appears to exist between crises and paralysis. In a number of cases unilateral or bilateral palsy of the abductors, or complete recurrent palsy, is met with without any previous crises; in a second series no paralysis ensues, even after occurrence of frequent and severe crises; whilst in a third series both spastic and parálytic phenomena coincide in the same case. Should palsies occur, the law of the greater vulnerability of the abductors holds good. The spastic phenomena are probably due to an increased irritability of the adductor centres (F. S.). A peripheral stimulus conducted along the centripetal fibres of the superior laryngeal to those centres which, according to this hypothesis, are in a condition of increased irritability, does not set up a mere cough, as under normal conditions, but spasmodic coughing, spasm of the glottis, general convulsions, in short a crisis. It also explains the influence of cocaine applications upon the larynx in laryngeal crises. The course of these palsies is generally slow and progressive, and the prognosis always unfavourable. The spasmodic attacks vary greatly in frequency. They may occur but two or three times in the course of years, or they may occur daily, or even two or three times a day. In some cases they occur spontaneously; they may come on suddenly during sleep, or they may be set up by slight forms of irritation, such as coughing, swallowing food or cold fluids, or on slight exertion. On the whole, it may be said that they belong to the early manifestations of the disease and tend to disappear in its later stages.

In patients subject to laryngeal crises it is most important to observe the greatest caution in taking food. As a laryngeal crisis may come on suddenly, food should always be minced, lest a mass become impacted in the glottis and drawn in during the long inspiration; although the initial phase of coughing, if it occurs, would be a safeguard to the patient. All sources of local irritation, such as the ingestion of cold or very hot food, should be avoided. A cocaine spray or a solution applied to the larynx will often cut short an attack or a series of attacks; and one of us (W. W.) has seen marked relief from the inhalation of nitrite of amyl.

In *labio-glosso-laryngeal paralysis* anaesthesia of the larynx has been observed, but laryngeal crises are almost unknown. In several cases weakness of the glottis-openers has been noted. One of us (W. W.) observed bilateral paralysis of the internal tensors alone, without any abductor paresis; although the other usual features in the tongue and soft palate were well marked. Permewan has observed complete recurrent paralysis within nine months of the commencement of abductor paresis.

In *disseminated cerebrospinal sclerosis*, laryngeal paralysis is very rare. One of us (W. W.) has observed tremor of the vocal cords on phonation, and coarse tremor on abduction. The slow monotonous tone, with jerky voice and scanning speech, is an early feature in most cases.

In *syringomyelia* both motor and sensory lesions, either unilateral or bilateral, are often present in the larynx; particularly the latter. Cartaz, analysing eighteen cases observed by French laryngologists, found that the larynx was involved in about 50 per cent of the cases. In some there was diminished tactile sensation in the larynx, amounting in a few to total anaesthesia; in others thermic sensation alone was affected.

Palsies of the muscles supplied by the recurrent laryngeal nerves have also been observed, the abductors failing first. In total paralysis of long-standing the vocal cord or cords are said to become atrophied.

Laryngeal crises do not appear to have been observed.

In *general paralysis of the insane*, Permewan concluded, from an examination of 34 cases, that in at least 20 per cent there is more or less marked abductor paresis. His observations again confirmed the general truth of the law laid down by one of us (F. S.) as to the special liability of the abductors to succumb in organic disease.

FELIX SEMON.
WATSON WILLIAMS.

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The enormous literature of this and allied subjects may be shewn by the large number of references, 1033, appended to an article by one of us (F. S.) on "Laryngeal Neuroses" in Paul Heymann's *Handbuch der Laryngologie und Rhinologie*, 1897. Hölder, Wien.

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F. S.
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THE NOSE, PHARYNX, AND LARYNX IN THE ACUTE SPECIFIC FEVERS.

DIRECT LARYNGOSCOPY, TRACHEOSCOPY, BRONCHOSCOPY, OESOPHAGOSCOPY, AND GASTROSCOPY.

FOREIGN BODIES IN THE AIR- AND UPPER FOOD-PASSAGES.

DISEASES OF THE TRACHEA.

THE NOSE, PHARYNX, AND LARYNX IN THE ACUTE SPECIFIC FEVERS

By P. WATSON WILLIAMS, M.D.

Introductory Remarks.—A priori, it would seem inevitable that the nose and throat should be the chief portals of entrance into the system of the specific air-borne infective organisms for most of the acute infectious fevers; whilst water-borne diseases, such as enteric fever, would only in much less degree be apt to infect the mouth and fauces. In 1897 Dr. de Havilland Hall stated that "evidence is gradually being accumulated which points to the conclusion that people who suffer from nasal affections are more liable to contract infectious diseases, especially diphtheria, than healthy persons. . . . If we except water-borne diseases, such as cholera and dysentery, almost all the other infectious diseases are conveyed by the air, and hence the morbific agent usually enters by way of the nasal cavities and pharynx, and here it proliferates during the first stage of incubation." That in the first stages of many acute specific fevers the nose and throat present early manifestations of considerable diagnostic value is commonly recognised, but it is desirable to accentuate the importance of regarding the upper air-passages as the usual site of the initial lesion, not perhaps because local treatment exercises any very definite influence on the clinical course of these diseases, but because (1) unhealthy conditions of the nose and throat render the individual more susceptible to infection; (2) actual breaches of surface, for example wounds or operative treatment, increase the risk of infection in those who are subsequently exposed to contagion; and (3) the specific organisms often persist for a considerable period, certainly in the secretions, and probably in the lymph-spaces and tissues of the nasal and faucial mucous membranes, thereby constituting the chief means of spreading the infection to others.

Without repeating the remarks on the part of the tonsil in health and disease (*vide* p. 162), it is opportune here to refer to the investigations of Jonathan Wright on the differences between the behaviour of dust and of bacteria in the tonsillar crypts. In imitation of the work of Goodall and others he dusted carmine powder on the surface or injected it into the crypts of tonsils, ten minutes or so before tonsillotomy, and subsequently examined sections of the removed tonsils. He found that the carmine particles did not pass readily through the epithelium, but were seen passing freely in limited areas only. There was a striking difference in the behaviour of the carmine from that of bacteria, both on the surface and in the crypts, for though all the carmine had been washed away by the technique of preparation for the microscope, not only from the surface but also from the crypts, streams of carmine particles that had at once penetrated could always be seen between the epithelial cells or in the lymph spaces, whilst, though the bacteria could usually be demonstrated in these same tonsillar crypts and along the surface, "they do not pass within, though they may have been there indefinitely." Yet when one of a pair of tonsils was pierced by a sterile stylet of small calibre, the pierced tonsil revealed small colonies of bacteria growing around the damaged areas at a distance from the epithelium. "This would seem to indicate that deep infection of the lymphoid tissue, even with surface bacteria carried in the stylet without local disturbance of tissue and without much resulting inflammation, meets with less resistance to growth than near the surface, even when the epithelium is partly removed. This is in direct accord with the idea that at the surface exists an adaptation requisite to meet the exigencies of habitual environment which do not exist more deeply, and that it is not so much the character of the tissue as its situation which counts in the function of resistance to infection, nor does so much depend upon the violence of the initial insult to the tissues as upon its depth." Such investigations indicate first, that a successful inoculation of the host is not so much due to a mechanical invasion of the tissues through the open spaces of the epithelial surface, which led Gerhardt to speak of the tonsil as a "physiological wound," as to a failure in resistance either from the virulence or overwhelming numbers of the invading organisms, and secondly afford an explanation of the increased susceptibility of the tissues of wounded surfaces. It is still an open question how far the nasal passages constitute the direct portal whereby pathogenetic organisms gain entrance to the higher nerve-centres. There is no room for doubt that in some cases the infecting organisms giving rise to intracranial abscesses or leptomeningitis obtain access to the cerebral meninges through the nasal passages (*vide* case on p. 287), but it is probable that some cases of cerebrospinal meningitis, in which the meningococcus has been obtained from the nasal passages, and some cases of tuberculous meningitis are similarly due to infection through the nose.

Chronic hypertrophy of the tonsils and adenoids results in the formation of masses of lymphoid tissue that have partially failed to resist the invasion of organisms; they not only lead to a lowering of systemic resistance, but they are indications of weak and damaged resistance to invasion, hence their presence constitutes a factor which cannot be ignored in the invasion, clinical course, and complications of many of the acute specific fevers.

Influenza is due to an infection which undoubtedly enters the system through the upper air-passages, and in a large number of cases characteristic symptoms and lesions are manifested in these regions. In many cases, however, from the beginning to the end of an attack of influenza, there may be complete absence of any nasal or pharyngeal catarrh, the whole brunt of the attack appearing to fall on the nervous system or the abdominal viscera. In the upper respiratory tract influenza produces (1) inflammatory affections, and (2) nervous lesions.

In the majority there is a greater or less degree of naso-pharyngeal coryza, and very frequently it culminates in an acute inflammatory affection of the mucous membrane of the nose and its accessory sinuses. The nasal or naso-pharyngeal catarrh at the outset is often indistinguishable from ordinary acute coryza, but early spontaneous epistaxis is common in influenza, and should therefore excite suspicion. Moreover, the severe course of the mucosal inflammation and the degree of submucous infiltration strongly suggest influenzal infection. The extremely common and acute brow-ache is due to acute inflammation of the frontal sinus, or even of the antrum. In this latter case the pain is usually mainly concentrated in the region of the implicated antrum, but often enough the referred pain over the corresponding supraorbital region suggests acute frontal sinusitis. The thickening of the inflamed mucous membrane is frequently sufficient to cause complete closure of the apertures of exit, consequently the copious muco-purulent secretion is retained till, with the ever-increasing neuralgic pain, which may become exceedingly acute, the contained secretion forces an exit; then with the gush of outpoured mucopus there is immediate relief to the pain. Very often this relief is only temporary, the same process of accumulation, followed by escape of retained secretion, recurs with diminishing severity once, twice, or oftener till the acute stage has passed. When the antrum is the seat of the collection the escaping fluid has often a greenish tinge, and is generally fetid, the secretion of a frontal or sphenoidal sinus being sometimes more distinctly yellow. When the sphenoidal sinus and the posterior ethmoid cells are affected the symptoms are the same, but the condition is more dangerous from the greater liability to secondary infection of the cerebral meninges; the specific infecting agent, or the pyogenetic organisms that flourish in the inflamed mucous membrane, gaining access to the base of the brain through the cribriform plate, or through the communicating lymphatics or veins. Although at present there is not conclusive evidence that direct infection of the meninges from the nasal passage is the usual course of events, this seems a priori to be the most direct and obvious route, and there is no doubt that, in some cases at least, meningeal infection has so arisen. Moreover, such infection from the nasal passages probably occurs in some cases without any symptoms pointing to intranasal disease of any kind. I have reported an instructive example of this sequence (19); a boy complained of acute pain in the right ear, photophobia, and slight febrile disturbance. The previous history suggested a mild influenzal attack, and, as the ear was normal, a diagnosis of sphenoidal sinusitis with meningeal irritation was arrived at. The cerebral symptoms developed into general cerebrospinal meningitis, the patient died, and, post-mortem, an accumulation of pus was found in the right cavernous sinus. It is probable that influenza infected the sphenoidal sinus, causing sphenoidal sinusitis and pain in the ear, and that the infection spread from the sinus to the cavernous sinus. Influenza causes more cases of acute and chronic sinus suppuration than any other infection; for the symptoms and treatment of these complications reference may be made to the section on Diseases of the Accessory Sinuses of the Nose (p. 72).

But less typical indications of acute infective inflammatory rhinitis may occur, such as painful or inflammatory oedema round the orbit. Dr. St. Clair Thomson records an instance in a boy of eleven in whom an abscess formed over the frontal sinus, and three days later there was tense oedema, without redness, of the right eyelids. No pus could ever be found in the nose, and yet, on incising the swelling there was an escape of free fluid pus, welling out from the right frontal sinus and containing the *Micrococcus catarrhalis* in pure culture. The boy had only "had a slight cold, was out of sorts, and complained of a sore throat." In other patients again no suppuration may appear at any period, and yet the eyelids may become very oedematous, or there may be simply a persistent boggy redness and thickening of the middle turbinal, or in the neighbourhood of the middle or posterior ethmoidal cells.

Acute naso-pharyngeal catarrh, or acute pharyngitis, is almost the rule and is often very painful, the pain darting into the ear and being associated with dusky red, boggy, or even oedematous thickening of the inflamed, sometimes haemorrhagic, mucous membrane; and not seldom the acute stage passes into a rather persistent subacute or a chronic pharyngitis, or naso-pharyngitis. The more than usually deep oedematous infiltration may extend to the underlying palatal and constrictor muscles, causing pain and difficulty in swallowing.

Many cases of influenza begin with acute tonsillitis, and it may not be possible to determine the influenzal character of the affection until by the spread of the contagion to other members of the household its true nature is revealed. It has been stated that in influenzal tonsillitis there is a tendency to the formation of a false membrane at and around the openings of the crypts, and that it may resemble a diphtheritic membrane by spreading over the tonsillar surface. I have never seen this.

The larynx and trachea very frequently are similarly affected in the acute catarrhal laryngitis and tracheitis, sometimes manifesting itself in a degree of oedematous swelling that results in aphonia or in laryngeal stenosis. Infraglottic swelling has often been noticed, and Fränkel has described a fibrinous infiltration of the vocal cords resulting in a whitish appearance in the affected regions of the cords, and superficial excoriations of the vocal cords may also occur.

In a few instances deep septic inflammation of the pharynx and larynx has occurred, the symptoms, signs, and clinical course differing in no respect from acute septic inflammation of these regions from other infections; it need hardly be said that these forms of influenzal throat complication are very dangerous to life.

Haemorrhage from the nose has already been mentioned as a frequent early symptom of influenza, but a similar tendency to bleeding is often noticeable in the naso-pharynx, pharynx, larynx, or trachea. The bleeding is often a capillary oozing, and sometimes if the bleeding area be carefully cleaned no bleeding point can be seen, the blood seeming to transude through the inflamed but intact mucous membrane. Similarly haemorrhagic laryngitis, or submucous laryngeal haemorrhage, may arise. Again, in the trachea similar oozings of blood have been seen.

The escape of blood often appears to relieve the engorgement of the region implicated, and is seldom in itself a matter of any gravity. But it may cause much anxiety, especially in cases in which the symptoms have not been recognised as a transient manifestation of influenza, and the suspicion of pulmonary tuberculosis may arise. The absence of any bleeding point, and particularly where the source of the oozing is laryngeal or tracheal, together with the diurnal rise and fall of temperature, which so frequently persist after even a mild influenzal attack, may for a time cause much difficulty in excluding the possibility of early pulmonary tuberculosis.

Neuroses.—The nasal neuroses that may follow influenza are neuralgia and anosmia. Neuralgic pains are most commonly referred to the root of the nose, but do not call for any special discussion. Anosmia is not very rare after influenza, but is generally temporary, seldom persisting more than a month or two, yet sometimes permanent. Perverted sense of smell is an occasional nervous sequel, but when cacosmia or subjective perception of a bad odour is complained of, or an intermittent disagreeable taste, careful search should be made for the existence of latent sinus suppuration.

In a few instances paralysis of the soft palate has followed influenza, but a rather persistent condition of hyperaesthesia is less rare. Kuttner observed paresis of the soft palate and muscles of the posterior wall of the pharynx on the left side and of the left vocal cord following influenza.

The most common laryngeal neurosis is paresis of the internal tensors of the vocal cords, probably in most instances a myopathic rather than a true neuropathic paresis. In a good many cases paralysis of one or both abductors of the vocal cords has been recorded, and even complete recurrent paralysis may follow influenza. These lesions are undoubtedly due to peripheral neuritis; sometimes the muscles quickly recover power, in other cases the paralysis persists for a long period or never clears up.

Enteric Fever.—The nasal passages seldom attract attention by notable symptoms, although epistaxis at the onset of the fever is common, and at this stage simple rhinitis, in greater or less degree, by extension to the Eustachian tube, may cause temporary deafness. Whether such rhinitis is directly due to the *Bacillus typhosus* is undetermined, but that the specific organism gains entrance to the nasal passages in some cases, seems clearly proved by the discovery of the bacillus in the pus of antral empyema following an attack of enteric fever. Nasal accessory sinus disease, however, is a rare complication of this fever. Both the pharynx and larynx are more or less affected in a considerable proportion of cases of enteric fever; indeed, some degree of pharyngeal catarrh and laryngitis is not uncommon in the earlier stages, though the more serious gross lesions of the larynx are later in onset.

Pharyngitis and laryngitis and various degrees of tonsillitis, follicular or parenchymatous, or a painful herpetic eruption on the pharyngeal mucosa, may be the most notable symptoms at the onset of the fever, and, particularly when associated with bronchial catarrh, may be misleading and obscure the diagnosis. Epistaxis, too, is not unusual at the commencement, and this, together with the angina and laryngitis, makes the onset hardly distinguishable from that of some forms of influenza, especially during the first two or three days of the fever. Otherwise these early throat symptoms have no prognostic importance. Later small necrotic areas like whitish specks sometimes appear on the fauces, tonsils, or pharynx, and breaking down leave superficial or deep ulcers; thus, Prof. Osler records a fatal case in which there was irregular ulceration, five centimetres long and two broad, on the posterior pharyngeal wall; such ulcers are usually round and oval, and, according to Murchison, from two lines to three-quarters of an inch long. In these areas the ulceration may be preceded by a patch of necrotic epithelium resembling false membrane; in other cases pseudo-membranous exudation occurs similar to that met with in scarlet fever. Somewhat similar superficial, clean cut, circumscribed ulcers have been observed on the soft palate near its junction with the hard palate. Although the condition has been described as pharyngo-typhoid, the ulcers are probably always due to streptococci or staphylococci.

Cancrum oris occurs occasionally, most frequently in children; beginning as a small necrotic patch on the inner side of the cheek and gradually spreading in depth, it extends to the external surface until, as the patch of gangrene separates, a perforation is formed.

The chief *laryngeal complications* are: (1) catarrhal laryngitis and oedema, (2) membranous laryngitis, (3) ulceration, (4) perichondritis, (5) paralysis. There is nothing characteristic about (1) *catarrhal laryngitis*, to which sufficient allusion has already been made. (2) *Membranous laryngitis* is rare, but occurs occasionally, apart from true diphtheria, which is a possible complication. The formation of false membrane is probably evidence of the intensity of the reaction to infecting organisms, and is liable to be accompanied by oedematous infiltration of the submucous tissues; hence, apart from the stenosis due to the membrane, the glottic aperture is diminished by the swollen mucous membrane. In the only instance in my personal experience, the dyspnoea necessitated relief by intubation, the false membrane coming away when the tube was coughed out. The laryngeal symptoms subsided before the death of the patient, a day or two later, with symptoms of intestinal perforation. Lewy records a case of "laryngitis fibrinosa," the child being tracheotomised on account of the resulting laryngeal stenosis, and after death there was found swelling of the mucous membrane, which formed a whitish swelling below, occupying the whole of the subglottic region.

(3) *Laryngeal ulceration* is by no means rare in enteric fever, and more systematic examination of the larynx would probably shew that the ulcers simply represent later stages of localised submucous infiltration. Pyogenetic organisms pass through the altered superficial epithelium and produce small abscesses, which become ulcers after disintegration of the superficial epithelial layers. The frequency of laryngeal ulceration in enteric fever seems to vary considerably, as might be expected in a disease which exhibits such wide variations in type in different epidemics and in different localities. The commonest sites of the ulcer are the tips and edges of the epiglottis, the neighbourhood of the vocal processes, and the posterior surface of the cricoid. The region of the vocal processes is subject to attrition in coughing and speaking, whilst the other seats of election for the ulcer are attributable to the action of food in swallowing. As to the nature of the ulcers, probably the great majority of instances are due to secondary infection by pyogenetic organisms (Kanthack and Drysdale), and not directly due to specific infection by the *Bacillus typhosus*. A certain number of the ulcers, however, are probably associated with the typhoid bacillus, particularly the deeper ulcerations, and these must be regarded as specific typhoid ulcers similar to those in the intestine (Landgraf). Apart from the symptoms and course of these laryngeal ulcers, it is evident that the occurrence of specific typhoid ulcers in the larynx must constitute a dangerous source of infection to those in attendance. Many years ago, Budd, physician to the Bristol Royal Infirmary, maintained that enteric fever was infectious (as well as contagious). This opinion is supported by some cases which I recorded in detail in 1894. A male patient was admitted to the Bristol Royal Infirmary on the sixth day of his illness; he became very delirious, and about ten or eleven days later had well-marked laryngitis and bronchitis, and was constantly coughing and expectorating about the bed. He died suddenly on the twenty-ninth day of the illness with symptoms of laryngeal obstruction. At the autopsy the epiglottis and arytaeno-epiglottidean folds were markedly oedematous, and the ventricular bands and the right vocal process much ulcerated, the ulcers being covered with purulent sloughy debris. The other lesions were characteristic of enteric fever. With every precaution against contamination, cultures were made in agar from the ulcers of the larynx, and demonstrated the presence of the *Bacillus typhosus*. Fourteen days after this patient's death one of the nurses in attendance on him shewed symptoms of enteric fever, which, after running a peculiarly virulent course, ended fatally. She had not been in

contact with any other case of enteric fever, and, of course, she had followed all the usual precautions in dealing with the patient's faecal and other discharges. Yet another patient, in another ward altogether, and the only one of the three under my care, began, about the same time as the nurse, to develop enteric fever, with headache, laryngitis, and bronchitis. He had been in the ward for some months under treatment for aortic aneurysm. On the ninth day of illness he became extremely cyanosed and died. At the autopsy, in addition to all the characteristic changes of enteric fever in the second week, there was superficial ulceration on the vocal processes of both sides, and on the arytaenoids. As there was no other case of enteric fever in the ward, and as he was rarely, if ever, visited by any friend from without, and had no opportunity of acquiring the disease through the ordinary channels of infection, it was difficult to account for his attack, until, on inquiry, I found that he was in the habit of visiting a patient in the same ward as the first patient who was proved subsequently to have ulcers with typhoid bacilli, which, through the expectoration so freely scattered about, infected both the nurse and the patient with aneurysm. Although laryngeal ulcers in enteric fever do not, as a rule, affect the ordinary course of the disease, the occurrence of much glottic swelling is exceedingly dangerous. Keen and also Marsden insist that in adults a marked degree of laryngeal stenosis may occur before signs of distress arise, and then the very first attack of dyspnoea may be unexpected, sudden, and fatal. After tracheotomy the patient very often recovers.

(4) *Perichondritis.*—In some cases the ulcers spread by sloughing and extend so deeply as to invade the perichondrium, and may thus induce necrosis of the underlying cartilage. The arytaenoid and the cricoid cartilages are those most often implicated, and the disintegration of the tissues may cause foul expectoration for a long period, till at length the necrosed cartilage is eventually exfoliated, or, as more often happens, death intervenes. In two cases observed by Prof. Osler, both patients recovered after expectorating large portions of the thyroid cartilage. Oedema of the laryngeal mucous membrane and tissues is very prone to arise in association with the ulceration.

(5) Nerve lesions in the throat due to enteric fever are rare, but *paralysis* of the vocal cords has been observed, and may be either (*a*) the result of toxic peripheral neuritis, or of a central nuclear lesion, being sometimes associated with peripheral neuritis elsewhere, or (*b*) due to pressure on the recurrent laryngeal nerve by enlarged glands. Abductor or complete recurrent paralysis may thus arise on one or both sides. These neural paretic affections usually appear late. Muscular paralysis may also occur as the direct result of inflammatory changes in association with acute oedema, ulceration, and so forth, and, as a consequence of perichondritis, especially in and around the crico-arytaenoid joints, ankylosis is apt to ensue, with fixation of the corresponding vocal cords.

Whooping-Cough. — In the prodromal stages a diffuse catarrhal condition of the upper respiratory tract is not uncommon, suggesting a bronchial cold with some subacute rhinitis and pharyngitis, but, with the appearance of the characteristic manifestations of the whoop, epistaxis commonly occurs and the prodromal catarrh often subsides or disappears, except that the spasmodic cough continues to be followed by the expectoration of small pellets of sticky mucus. In ordinary attacks the larynx shews little except some catarrhal inflammation of the arytaenoid and inter-arytaenoid regions; occasionally laryngeal oedema results from the violent coughing. In the most severe attacks in patients with general nervous manifestations, the laryngeal spasms may be so severe and prolonged that no air can be inspired, and convulsions, ending in death, may occur. Less severe attacks of laryngeal spasm with carpo-pedal contractions may culminate in sudden death, the symptoms being practically identical with the more severe forms of laryngeal vertigo. The laryngeal stenosis with inspiratory whoop, so characteristic of the affection, is a neurosis, due to adductor spasm, and is probably purely central. The bulbar centres appear to retain their irritable and unstable condition for some time after the attack has subsided; and even when the laryngeal spasms have disappeared for some weeks or months, a catarrhal laryngitis arising in the course of an ordinary cold, or cough excited by any peripheral irritation in the larynx, is often attended by a spasmodic inspiratory whoop.

There do not appear to be any characteristic or constant laryngeal changes in whooping-cough, but Herff, who examined his own larynx during an attack, observed an initial slight catarrh which became intense during the spasmodic stage, and with each paroxysm a pellet of mucus appeared on the posterior laryngeal wall, the paroxysms ceasing when this was expelled.

Patients who have been exposed to the infection, but who have previously had whooping-cough, may simply suffer from recurring laryngeal spasm, without any other symptom, and the real nature of their affection, which is often most alarming to the patient, is liable to be mistaken for something much more serious.

Small-pox.—Epistaxis is not common in the early stage of small-pox except in the malignant forms. Pharyngeal and laryngeal catarrh is usual in most cases of small-pox, and in the majority some vesicles appear on the fauces, and in the larynx. Before any vesicles appear, the pharyngeal and laryngeal mucous membrane is usually dusky red, and the inflammation may be so severe as to cause much swelling of the fauces, the uvula, and the tonsils. The pustules have been observed in the nostrils as well as in the pharynx and larynx. In the latter situation they cause more or less severe laryngitis about the sixth day, but by the ninth to the twelfth day graver symptoms often arise, due to oedema of the affected laryngeal mucosa around the pustules, or even more frequently to pseudo-membranous laryngitis. The formation of false membranes generally comes on, according to Ruhle, about the tenth day. The vesicles are seen in the larynx as discrete, well-defined white spots, standing out from the surrounding mucous membrane on the epiglottis, arytaenoid regions, and on the vocal cords. The epithelial covering of the vesicles soon becomes denuded, leaving excoriations or superficial ulcers. Secondary inflammation of the fauces and adjacent parts may follow, with cervical adenitis, which may go on to suppuration. Interstitial laryngeal haemorrhages and membranous laryngitis are not uncommon complications of malignant haemorrhagic small-pox. With any of these complications the disease may run a rapid course, either ending in death or resulting in cicatricial contraction of the ulceration and laryngeal stenosis. Perichondritis and muscular laryngeal paralysis are also prone to arise. In some cases in which ulceration extends down to the cartilages, necrosis is likely to result with extension of the dead cartilage, or chronic contraction with progressive stenosis for years may ensue. Dr. de Havilland Hall mentions a patient on whom tracheotomy had to be performed thirty-eight years after the attack of small-pox, and during the whole of this period she was the subject of a chronic laryngeal affection. The earlier throat manifestations of small-pox are very similar to those of measles, and are liable to be mistaken for those of that disease, particularly when there is some coryza and lacrymation, symptoms strongly suggesting the onset of measles.

Chicken-pox.—The vesicles may often be seen on the fauces, lips, tongue, and cheeks, and rarely in the larynx at the same time as the skin eruption. Rondot has observed a varicellous eruption on the buccal mucous membrane even in the absence of a cutaneous eruption, in the course of an epidemic. They generally soon lose their epithelial covering and form superficial ulcers. The throat affections of varicella are, however, with very rare exceptions, unimportant. But with laryngeal vesicles the resulting laryngitis in young children may necessitate tracheotomy, and Rogers and Bayeux describe a fatal case of haemorrhagic chicken-pox in an infant, aged six months, suffering from progressive dyspnoea. At the autopsy it was found that the edge of the epiglottis was gangrenous, and that there was a strip of slough on the free borders of the vocal cords, a crateriform erosion of the left vocal cord, and a varicella spot on the mucous membrane of the left pyriform fossa (*vide* also Vol. II. Part I. p. 481).

Scarlet Fever.—The most constant symptom of this disease is the throat affection; in fact, a greater or less degree of inflammation of the faucial mucosa is probably the one symptom never absent in the earlier stages of this exanthem, and there is some reason to believe that the throat is a not unusual site of primary infection. The scarlatinal throat generally shews a characteristic bright-red injection of the fauces and tonsils, associated with a reddening and enlargement of the papillae of the tongue. Fever, nausea, and vomiting, when accompanied by these conditions in the throat, should always excite suspicion, and a search for the rash of scarlatina, which, however, may not have appeared, or may have been so slight and fleeting that the diagnosis must be made from the throat manifestations alone. Greater difficulty in determining the true nature of the throat affection arises in patients who, having previously suffered from scarlatina, have again been exposed to infection. In these more or less immune individuals general systemic disturbance may be absent or very slight, the only complaint being of a sore throat, and the appearances those of simple tonsillitis, with a slightly membranous or sloughy appearance of the swollen faucial mucosa and some enlargement of the cervical glands. These cases are dangerous, for the mild character of the disease often leads to their true nature being overlooked; and, as the patients are capable of conveying the infection, the results may be disastrous not only to others but to themselves, as, from undue exposure without any adequate precautions, severe and destructive otitis or other complications may arise.

The degree of throat inflammation varies in scarlet fever from the slightest reddening of the fauces to the most severe membranous or gangrenous inflammation with secondary implication and suppuration of the deep cervical tissues, and may be conveniently classified in three groups, namely: (*a*) Simple angina, redness with slight infiltration of the submucous tissue of the fauces, soft palate and uvula, and posterior pharyngeal wall, with parenchymatous or follicular tonsillitis. (*b*) Membranous angina, a pellicle forming on the intensely inflamed and swollen tonsils and fauces, and extending in some cases to the larynx. There is more or less swelling of the connective tissue of the submaxillary region. The false membrane generally appears about the third to the sixth day of the fever, and is due to streptococcic invasion of the epithelium and deeper tissues. The cases are usually of a severe type, and superficial ulceration of the tonsils and fauces is common. The chief cause for anxiety is the differentiation from true diphtheria, as diphtheria is not uncommonly a complication of great danger in a scarlatinal patient. A pure streptococcic false membrane may generally be distinguished from true diphtheria by its being yellowish in colour and more friable, instead of greyish-white and tough. As a general rule diphtheric symptoms appearing in the course of the fever are not due to the Klebs-Loeffler bacillus; on the other hand, similar symptoms occurring as a sequel to scarlatina, that is, in two or three weeks from its commencement, and very rarely before the second week, are usually due to true diphtheria. (*c*) Phlegmonous angina, Scarlatina anginosa. The tonsillar and faucial inflammation is a marked feature in these cases, either from the first, or, even more frequently, when the fever reaches its highest point, or during the subsidence of the rash. The throat condition resembles those of acute septic pharyngitis and laryngitis, the mucous membrane being much swollen and oedematous, with a somewhat livid or purplish hue; the uvula is often swollen to an enormous size and is club-shaped. The submaxillary region is generally greatly swollen and brawny, the phlegmonous inflammation of the connective tissue often spreading under the jaw and down the neck. Swallowing is difficult and very painful, and viscid mucus and mucopus collect in the mouth and throat. The inflammatory process is also apt to spread to the larynx, producing laryngeal oedema with hoarseness, aphonia, or acute dyspnoea; to the posterior pharyngeal wall with swelling of the mucosa and severe retropharyngeal abscess. Not only are the faucial tonsils enormously swollen, but the rhino-pharyngeal tonsil is often similarly affected, the Eustachian orifices are swollen, and the middle ear becomes the seat of acute and destructive purulent otitis. The nasal mucous membrane, too, is prone to be acutely inflamed, and a copious muco-purulent secretion pours from the more or less blocked passages. The acutely inflamed areas are liable to early ulceration. Necrosis, deep ulceration of the tonsils, faucial pillars, or even of the soft palate, laryngeal perichondritis with necrosis of the cartilages, especially of the arytaenoids, suppuration of the parotid or submaxillary glands, or of the brawny swelling beneath the jaw, or other accessory sinuses of the nose, are amongst the complications encountered in these severe cases. Perichondritis of the laryngeal cartilages, when it does occur, is very liable to suppurate, and to be followed by necrosis (Kraus); whilst there is always the danger of further complications in connexion with the kidneys, heart, and other organs.

Treatment.—The general treatment of scarlatina and its complications does not come within the scope of these notes on the throat complications, but with regard to scarlatinal anginosa, it may be observed that (*a*) it is the general symptoms rather than the special character of the throat condition that, *in the earlier stages*, give warning of the severe nature of the case; (*b*) that whilst there is sometimes much dyspnoea, which may even cause asphyxia when the faucial or laryngeal inflammatory swelling is severe, the greater danger is more often in the liability to profound disturbance of the nerve centres or to cardiac failure. In the less severe cases, simple, soothing, and antiseptic gargles and sprays are useful; for example, permanganate of potassium, sanitas, or biniodide of mercury (1 in 4000) solution. When ulceration has occurred, local sprayings with weak solutions of biniodide of mercury, lotio nigra, of protargol, or argyrol, 8 to 10 per cent solution in water, are beneficial. Hot fomentations may be applied to the swollen neck and under the jaw, but ice and cold applications are possibly directly harmful to tissues of lowered vitality, which are therefore liable to undergo necrosis.

German Measles.—The fauces and tonsils are almost invariably diffusely red and sore in the initial stages of German measles, often resembling very closely in appearance and symptoms the less severe sore throat of scarlet fever. The solitary papillae of the tongue are sometimes distinctly enlarged and bright red. In very slight and mild cases there may be no sore throat and no febrile disturbance, but even in these there is seen the diffuse cherry-red hyperaemia. As a rule, the tonsils are red and swollen, and there is some pharyngitis, and almost always considerable swelling and tenderness of the posterior cervical glands. The redness may have a streaky distribution in the fauces and

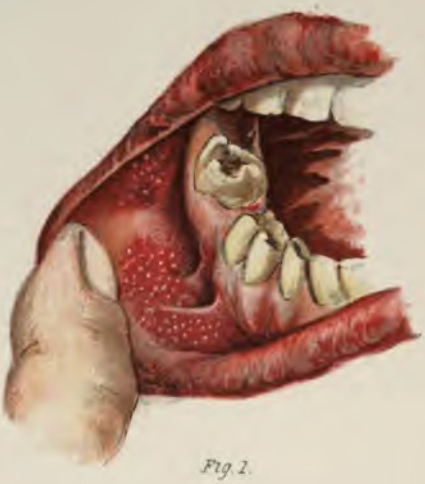

Fig. 1.

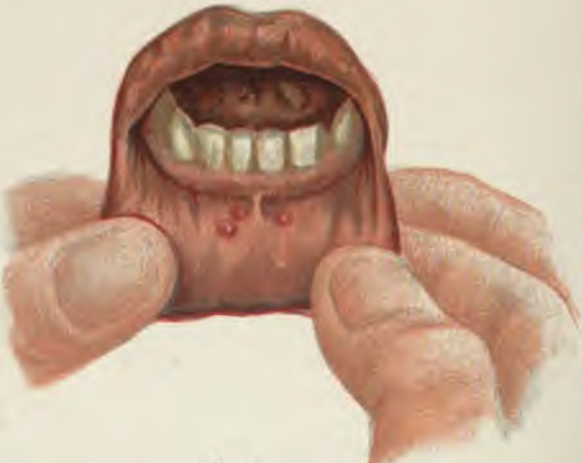

Fig. 2

PLATE XII.

"KOPLIK'S" OR "FILATOW'S" SPOTS,
the pre-exanthematous Sign of Measles.

Fig. 1. The early stages of typical appearance.

,, 2. The appearance of the spots at a somewhat later stage.
(Cases of P. W. W.)

soft palate; more frequently it is diffuse, but it lacks the patchy granular appearance of measles, even in those cases which develop into the morbilliform as distinguished from the scarlatiniform type of German measles. Not only the fauces, but the mucous membrane of the lips and the buccal mucosa often become a cherry-red colour, which often persists after the rash has disappeared. Such slight distinctions, however, are not sufficient to differentiate German measles from scarlet fever. In German measles the throat never shews changes comparable to those seen in scarlatina anginosa, and false membranes and deep ulcerations are unknown.

In what has been called the "*Fourth Disease*" (*vide* Vol. II. Part I. p. 448) there are no characteristic throat conditions apart from those described in German measles or the milder manifestations of scarlatina.

In **measles** some degree of pharyngitis, and especially laryngitis, is present in almost every case, but the larynx is usually more affected than the pharynx and the tonsils. The throat symptoms are seldom severe, and still more rarely are they the cause of anxiety. Nevertheless, the laryngitis may be attended with so much oedematous infiltration that urgent or even fatal dyspnoea may arise.

Whether there is a characteristic prodromal rash or endanthem in the fauces is a moot-point, but at any rate the soft palate and posterior palatal folds are usually congested, and there is a mottled, punctate, or "granular" rash on these parts and on the tonsils, giving a stippled redness; these appearances have also been observed on the laryngeal mucous membrane, especially of the vocal cords, ventricular bands, and inter-arytaenoid space. These changes may confirm a suspicion of measles aroused by the general prodromal symptoms, but I have never seen any manifestations in the throat which are, in my experience, characteristic of measles.

The most valuable confirmatory sign of measles, before the rash appears on the skin, are the small irregular spots of a bright-red colour with a minute bluish-white speck in the centre of each spot. These were originally described by Filatow, and Koplik was one of the earliest to emphasise their importance and value. They appear on the buccal mucous membrane, near its junction with the gingival mucous membrane, and also on the gums (see Plate XII.). They vary in number from two or three to several hundred, and are invariably discrete. They appear from about twenty-four hours to three or even five days before the characteristic exanthem, and begin to fade as the general eruption reaches its height. They must not be confused with the little milk-white specks of secretion which are sometimes seen in the same regions in any febrile condition; the specks of secretion are easily wiped off, the Koplik spots are not. I have often searched in vain for these spots in cases of measles occurring in epidemics, but I have never found them in cases which did not have true measles. Thus, the discovery of these spots appears to be of diagnostic value, but their absence is not decisive one way or the other. (See also art. "Measles," Vol. II. Part I. p. 388.)

Spasmodic laryngitis or false croup is fairly common in the earlier stages of measles in young children. Membranous laryngitis may occur in measles, but generally it comes on later, as the rash is declining. It is a dangerous complication. Faucial ulcers are very unusual, but laryngeal ulceration is not infrequent in severe cases, being commonly seen on the vocal processes or posterior laryngeal wall, and is probably caused by attrition from coughing. Instances of deep ulceration of the vocal cords and in the region of the arytaenoid cartilages have been recorded by Fränkel, who stated that they were due to pyogenetic cocci invading the tissues. Major has recorded a case of unusual severity with early aphonia, the left arytaenoid cartilage being found on laryngoscopic examination swollen and fixed in the position of abduction.

Paralytic sequels are very rare in measles, except the myopathic paresis of the internal tensors of the vocal cords, which is so often a temporary result of acute laryngitis.

In poorly nourished, unhealthy children gangrenous laryngitis, generally associated with gangrene of the mouth, may occur, the prognosis in such cases being very grave. In less severe attacks the discharge of the initial rhinitis may persist after the subsidence of the fever, and be the commencement of a chronic purulent rhinitis. The treatment of the throat complications of measles is the same as for similar affections due to other causes.

P. Watson Williams.

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P. W. W.

DIRECT LARYNGOSCOPY, TRACHEOSCOPY, BRONCHOSCOPY, OESOPHAGOSCOPY AND GASTROSCOPY

By Ernest Waggett, M.B.

Direct Laryngoscopy connotes inspection of the larynx without the aid of a laryngeal mirror. The method devised by Kirstein in 1894 has for many years found a recognised place in practice. Its special advantages over the indirect method are that the posterior wall of the cavity of the larynx comes fully under inspection, and that since the larynx is fixed and the observer's eye is brought in close proximity to it, topical applications can be made with great precision, and new growths and foreign bodies easily removed. The sole disadvantage is the unavoidable discomfort to the patient, though with proper cocainisation the procedure is quite painless.

Instruments.—Kirstein's original instrument has been superseded by the tube-spatula, which has been devised during the evolution of the method of bronchoscopy, but his illuminating apparatus with slight modifications still holds its place. It consists of a small electric lamp fixed, by a couple of universal joints, to a plate held upon the examiner's forehead by a strap or metal spring. It is enclosed in a cylindrical box capped by a plano-convex lens, adjusted to direct a pencil of parallel rays vertically downwards upon a small mirror fixed at an angle of 45°, which is pierced with a small central orifice. This is mounted upon a shield also perforated. These two perforations, in the shield and mirror, must, of course, coincide with the line of vision; and since the holes are necessarily small, they must be brought as close as possible to the examiner's eye; a want of appreciation of this small detail is the commonest cause of failure in those unused to the method. As the shield is liable to become heated, its lower edge, which must rest upon the skin of the cheek, should be covered with fibre. The examiner should not begin until he has definitely arranged the apparatus so that the line of vision and the line of illumination exactly coincide. The

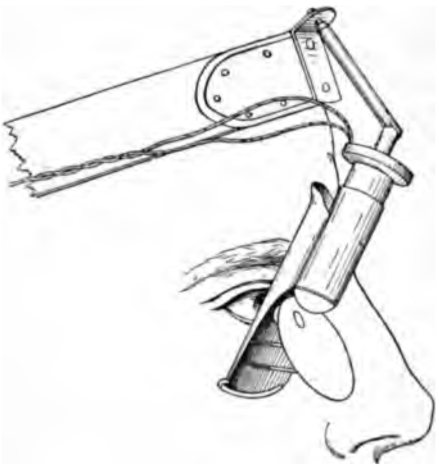

Fig. 83.—Kirstein's lamp in position close to the right eye. The large mirror enables a second person to obtain a reflected view, but not when narrow tubes are in use.

right eye is usually employed, and it should be protected from sputa by the use of a glass. Kirstein's lamp or Casper's prism lamp, in which the rays are diverted by a prism instead of a mirror, may be fixed upon the handle instead of the head; Brüning has devised such an arrangement with the addition that the lamp may be withdrawn some 3 or 4 inches from the tube-spatula in order to facilitate the manipulation of forceps. Guisez's head-lamp, consisting of three small converging bull's-eye lamps arranged round the eyelids, gives a good light. The tube-spatula has already been modified several times. An extremely convenient form is that devised by Killian; it is made to separate into two halves, so that they may be removed after a bronchoscope has been inserted through the glottis. The broad flat lower extremity is very serviceable in keeping the sides of the epiglottis out of the field of vision. Jackson has devised an instrument of somewhat similar form with a small lamp inserted at the laryngeal end, and with a rectangular handle which gives a mechanical advantage to the grip of the operator. The last feature is also adopted in Brüning's instrument.

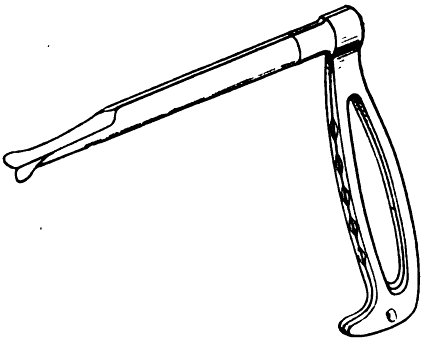

Fig. 34.—Killian's split-tube-spatula, separable into two halves after introduction of bronchoscope.

Technique.—Attention is here directed to the less expensive instruments, namely Killian's tube-spatula and Kirstein's forehead lamp, and mention will be made of swab-holders and forceps. Killian advises that when the air-passages are irritable, or the patient is nervous, morphine (gr. $\frac{1}{6}$) should be given to an adult, or a small dose of codeine to a child four hours before the operation. The pharynx, larynx, and especially the posterior surface of the epiglottis are thoroughly anaesthetised with a 10 per cent solution of cocaine with adrenalin added in order to prevent, as far as possible, general absorption of the former drug. An adult of ordinary mental composure can easily be examined by the surgeon single-handed, but in difficult cases success very largely depends upon carefully organised assistance. In order to save time, the method employed in the most difficult kind of case will be described; and the

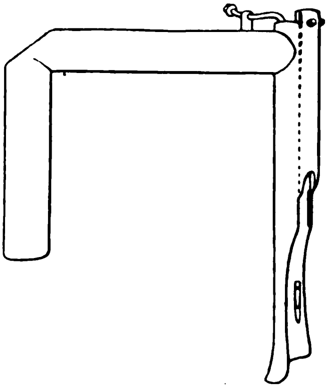

Fig. 35.—Jackson's tube-spatula with inset lamp and slide for removal after introduction of bronchoscope.

reader can then judge what will obviously be unnecessary in simpler cases. Thus, in the case of a child with dyspnoea, in whom a view of the larynx has not been obtained by the indirect method, it is possible that a large foreign body or new growth may be present, and consequently arrangements should be made so that tracheotomy, if necessary, may be done in an orderly manner. In one such instance I found it a great advantage to have had the trachea previously exposed. Where tracheotomy is at all likely to be needed, chloroform anaesthesia should be employed, and should generally be used for small children, in whom the emotional element and the nerve shock due to terror should be taken into consideration as important surgical factors. Laryngoscopy may be performed in the sitting posture even under chloroform, but I am of opinion that in all cases, whether under cocaine or under chloroform, the manipulations are very much easier when the patient is placed upon a table in the lateral position. The anaesthetist should confine his attention to the anaesthetic, make it his business to inform the surgeon of untoward changes, and be prepared to work in a darkened room. Two assistants are necessary; the first, seated on a stool close to the table on the left of the surgeon, is solely responsible for the position of the patient, who is drawn over the end of the table until the head and neck are free of it. The first assistant steadies the patient's head in the palm of his right hand, supporting the weight upon his right knee, the leg being raised by means of a stool. The left hand is free and may sometimes be used to hold a gag in the patient's mouth, passing under the neck to do so. In prolonged cases the first assistant's office is very arduous; he must be careful to get his leg support correctly arranged, otherwise his knee will tremble and cause embarrassment; he must also maintain the patient's body in the lateral position by packing pillows behind the back. The second assistant stands on the right of the surgeon, and is responsible for the preparation of a dozen swabs of absorbent wool securely fixed, and if he employ a nurse to prepare fresh ones, he must satisfy himself that they are secure and not leave this duty to the surgeon. In the case of the air-passages it is of the utmost importance to have absolutely secure swabs; the ordinary split-catch with a sliding ring is probably the best form, but as usually sold, the ring is apt to hitch against the end of the bronchoscopy tube and to cause a momentary delay when it is least acceptable. The proximal end of the ring should therefore be bevelled off carefully. All swabs and other instruments must be laid parallel on a roomy table so that they may be picked up quickly, and without confusion in the obscurity of the darkened theatre. After use, they should be taken from the surgeon by the second assistant in his left hand, while with his right he

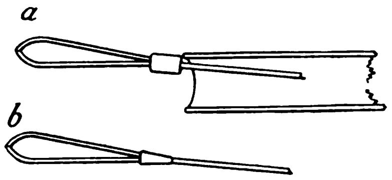

FIG. 86.—Wool swab-holders. (*a*) Incorrect, catching on end of bronchoscope; (*b*) correct, with collar bevelled.

hands up fresh ones held in the direction in which they will be used. He should also sponge the forehead mirror and lens when they become obscured by expectorations.

The surgeon sits on a low stool at the patient's head, his eye on a level with the latter. Regulating the lamp by means of a rheostat connected either with the street current or a secondary battery, so that it gives a full light without over-running, and having previously ascertained that he has spare lamps at hand, he arranges the apparatus so that the line of vision and of illumination exactly coincide, and directs the first assistant to retract the head until the upper incisors come almost into line with the axis of the trachea. A gag, when used, should be placed in the lower angle of the mouth and held, as previously described, by the first assistant. The surgeon takes the handle of the tube-spatula in his left hand, gripping it firmly between the fingers and palm, the back of the hand upwards, his forearm crossing over the patient's face; he then passes the flattened extremity back along the tongue, looking through the tube as he does so. The epiglottis is easily seen, and the instrument is then passed behind it and downwards for about ¾ of an inch. Now by a twist of the wrist, and taking care not to use the upper teeth as the fulcrum of a lever, he brings the tube as near as possible into line with the tracheal axis. This manoeuvre is, however, not only one of rotation but also of traction, for the extremity of the spatula must be made forcibly to pull forward not only the epiglottis, but also the hyoid bone and base of the tongue. It is very important to avoid plunging the instrument deeply into the pharynx, for when this is done it passes much more readily than is expected down below the arytaenoids. This is the common error with beginners. If the surgeon find any difficulty in recognising the parts under inspection, he may be fairly certain that something is wrong, for when properly introduced the instrument gives not only an unmistakable view of the organ, but a much more brilliantly illuminated one than that seen in the ordinary indirect laryngeal picture. With his right hand the surgeon uses swabs, probes, and forceps with the greatest ease and precision upon an operative field which is practically immobile and within a few inches of his eye. Should dyspnoea come on from glottic spasm, a bronchoscope can be passed through into the trachea. Bleeding after removal of growths may of course obscure the view and prevent further manipulations.

I have never seen any serious sequels, such as oedema, even in young infants, and the procedure is less severe than intubation. It is indicated as the routine method for removing papillomas in children. Numerous patterns of forceps have been devised for the removal of foreign bodies and of new growths. It is essential that if they are of the tube-forceps type, the movement shall be such that the jaws are not withdrawn into the tube, and away from the object. A long slender modification of the nasal punch-forceps introduced by Dr. Paterson will be found very serviceable for most cases.

With adults cocaine anaesthesia alone is necessary. Though the right lateral position gives a certain stability to the patient as well as a sense of confidence, the manipulations can be carried out very satisfactorily in the sitting posture, the patient being placed on a low stool. The first assistant stands behind him giving support to his back and firmly holding his head in an extended posture. The second assistant is on the right of the surgeon, who stands in front of the patient bending his face forward over the tube-spatula. Blood and secretions fall into the trachea and give rise to coughing. As opportunities for practising bronchoscopy are by no means common, and as the prone position is usually necessary, the surgeon should practise the manipulations on his laryngeal cases .in the lateral position. The method is difficult in nervous adults who cannot bring themselves to relax the muscles; and it is conceivable that cases will occur in which partial fixation of the jaw will render it impossible. I have, however, operated under chloroform upon an adult in whom the movements of the lower jaw were very much restricted, and was able to do so because the hyoid bone can be pulled forward to a far greater extent than would a priori be expected. To do this by leverage at the expense of the front teeth means faulty technique. Recognising the importance of this point, Chevalier Jackson has, as mentioned above, devised a tube-spatula with a rectangular handle, which when used in the sitting posture gives the surgeon a very full command in retracting the hyoid; in common with all the endoscopic instruments employed by Einhorn, Fletcher Ingals, and this surgeon, the illumination is supplied by a small lamp, inserted in a depression at the distal end of the instrument. The light, though very white, is not so powerful as that obtained in the larynx by the Kirstein lamp, and it may become obscured by blood and mucus. The rectangular handle is adopted by Brüning, whose new electroscope is, I understand, now usually employed in Killian's clinic.

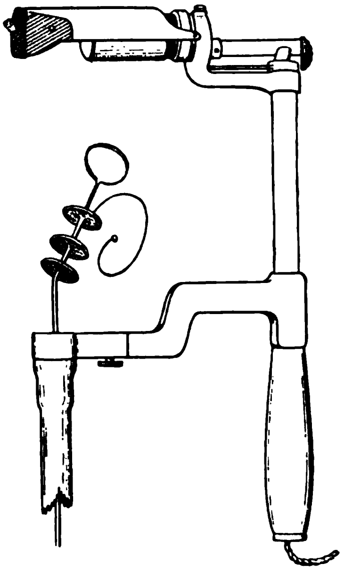

Fig. 37.—Brüning's electroscope, elevated from the handle, for use of forceps (Brüning's telescopic pattern with lock-nut).

TRACHEOSCOPY AND BRONCHOSCOPY naturally followed Kirstein's method of laryngoscopy, and any lengthy discussion as to the disputed question of priority would be out of place here. Von Hacker, von Mikulicz, and Kirstein examined the trachea in 1892, 1896, and 1897 respectively, but Killian first removed a foreign body from the bronchus in 1897, and there seems little doubt that he first realised the possibility of straightening out the left bronchus with a rigid tube. Since that year about 200 cases of foreign bodies alone have been reported.

Anatomy.—The method of bronchoscopy, which naturally includes tracheoscopy, owes its existence to the realisation that it is possible to pass a perfectly straight and rigid tube through the glottis into the trachea, and on into the main bronchi, and even into their main subdivisions, without doing any harm ; strictly speaking, this refers only to superior bronchoscopy, as inferior bronchoscopy connotes the introduction of the tube through a tracheotomy wound. Since in the employment of all endoscopic instruments the tube selected for any given case is the shortest and the largest which will prove serviceable, a few anatomical details must be considered. The length of a trachea can be ascertained approximately by measuring the distance between the

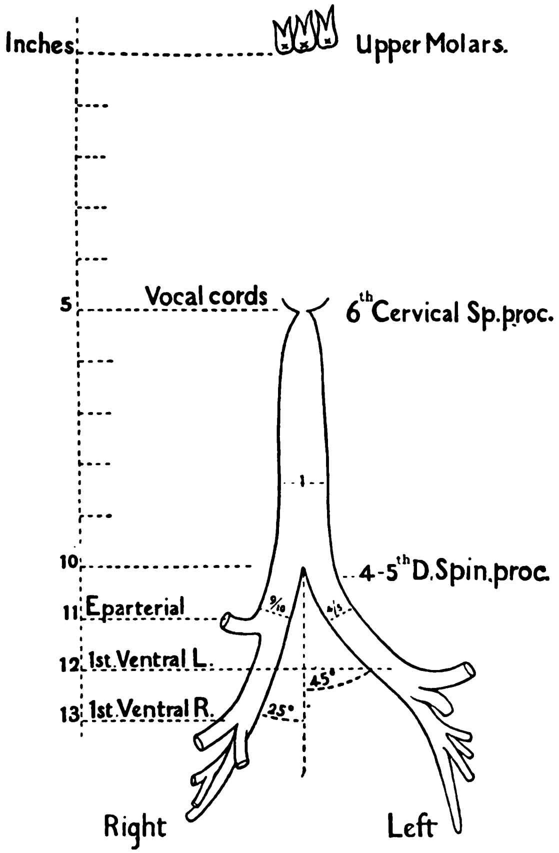

FIG. 38.—Scheme shewing average measurements in adult males from the upper molar teeth to bifurcation of the trachea, eparterial branch, first left and first right ventral branches (from data of Hasse, Sahli, Gottstein).

6th cervical and the division between the 4th and 5th dorsal vertebrae. In an adult man this is generally about 4½ inches (11 cm.), the distance from the glottis to the upper molar teeth being about the same. A tube, then, of 9 inches (22 cm.) will usually reach the bifurcation, but it may fall short of it by almost 2 inches in many individuals of quite ordinary stature. The diameter of the trachea may measure nearly an inch (22·5 mm.) in adult men, but ⅗ of an inch (16 mm.) is not uncommon both in men and women, whilst that of an infant a year old may be only ⅕ of an inch (5 mm.), and of a child of ten twice that measurement. The chief importance of these diameters is in connexion with performing inferior bronchoscopy in which tubes much larger than the larynx can accommodate may be used. For superior bronchoscopy, Killian recommends the use of tubes of 9 mm. and 7 mm. diameter for adults and children respectively, though a tube of 11 mm. can often be tolerated by adults. This is not surprising seeing that the adult male glottis is fully 20 mm. long, a measurement to be remembered in estimating the possible size of a foreign body. The trachea is not cylindrical but somewhat flattened from side to side. This may be important in reading a skiagram, for a flat foreign body seen lying in the sagittal plane is in the trachea, whilst one lying in the frontal plane is probably in the oesophagus. The posterior wall of the trachea is of course composed of soft tissue only; it is in relation with the oesophagus, the left posterior aspect being close to the descending aorta. The anatomy of the bronchi has been well described by Gottstein (16). The carina or division between the two main bronchi corresponds to the interval between the 4th and 5th dorsal spinous processes. It is often to the right of the middle line of the body, but in the majority of cases it is to the left of the tracheal axis, and as a consequence the right bronchus may seem to be almost a continuation of the trachea. Foreign bodies dropped into the trachea of the cadaver fall into the right bronchus, and in clinical practice enter the right more frequently than the left bronchus. This is partly because the right diverges from the axial line at an angle of about 25 degrees, whilst the left, on account of displacement of the lung by the heart, diverges at 45 or even 60 degrees. The diameters of the main bronchi are very nearly equal to that of the trachea, Hasse giving 23 mm. for the right, 20 mm. for the left, and 24 mm. for the trachea. Key and Brown, writing in 1831, made the pertinent statement that a shilling will enter the right but not the left bronchus. It will be remembered that the right bronchus differs from the left in giving off the eparterial branch (namely, one above the level of the pulmonary artery) to the superior lobe, which leaves the outer wall of the main stem at a distance of 1 inch from the carina. The left bronchus continues unbranched and with an undiminished diameter of 20 mm. for at least 2 inches in adult man, and sweeps outwards in a slight curve under the arch of the aorta and behind the left pulmonary artery. Four ventral and four dorsal branches are described, the first large ventral branch supplying the whole upper lobe. On the right side similarly about four ventral and four dorsal branches are given off, the first ventral supplying the whole middle lobe. Finally on both sides a continuation of the main bronchus may be traced right down to the bottom of the lower lobe. For surgical purposes the left bronchus may be divided by the site of origin of the first ventral branch, into a broad upper portion, 2 inches (5 cm.) in length, and a long narrower continuation with the orifices of some seven branches dorsal and ventral; the right bronchus is divided into three sections, a broad upper portion 1 inch in length, a middle narrower portion nearly 2 inches in length, terminated by the orifice of the first ventral branch, and a third long narrow portion with the orifices of some seven branches. It is important to get a clear conception of these anatomical details, as ordinary dissection gives a greatly distorted picture of the bronchial tree. The direction of the air-passages is best studied by taking skiagrams of the chest after injecting an emulsion of bismuth in oil into the intact cadaver, which is then rolled about (E. Goldmann). When mercury is used the parts are distorted by its weight. The calibre of the air-tubes is smaller in life than in the cadaver, and the measurement varies with the different phases of respiration, increasing to a recognisable degree during inspiration.

Inferior Bronchoscopy.—In the absence of abnormal secretions inferior bronchoscopy is a simple matter. When tracheotomy has to be performed for the removal of a foreign body, and experience has shewn that it should be done when a body of large size or of soft and friable nature is present, the operation should be done by the bloodless method. After incising the skin, the knife should be laid aside and the remainder of the dissection performed by means of two double blunt-hook retractors. The prongs should be made to overlap somewhat when introduced into the tissues of the middle line. Four or five retractions will generally expose the trachea, and the whole operation can, even in a small child, be performed quite bloodlessly in about two minutes. It is occasionally necessary to make a snip with the scissors to free the thyroid isthmus from its attachment above, and care must be taken in doing this not to wound any veins. The isthmus is divided between two Spencer Wells's forceps, which are thrust downwards to grasp not only that body but the venous plexus around it. A fruitful source of haemorrhage and delay is the snipping of the vein-traversed tissue below the isthmus; consequently this part should be divided by inserting the scissors from below upwards. The trachea should be freely opened, and its cut edges attached by a couple of stitches to the skin. Antiseptic gauze should be packed into the lower part of the wound and the stumps of the isthmus ligatured. A large-sized short unfenestrated oesophagoscopy tube 14 mm. in diameter may easily be inserted into one bronchus without embarrassing respiration by the other. Coughing must be prevented by painting the trachea, and especially the bifurcation, with adrenalin and cocaine, and the inflamed area in the neighbourhood of a foreign body requires several applications of a 10 per cent solution of cocaine. The instrumentation will be more fully described on p. 315.

Superior Bronchoscopy.—*Instruments.*—The simplest form of bronchoscope, that used until recently by Killian, consists of a long, narrow, cylindrical metal tube with a mat interior surface and marked in centimetres externally. The proximal end widens to a funnel shape, and a strong lateral projection enables it to be fixed in a universal handle. About 7 cm. from its distal end it is pierced by a couple of perforations 2 cm. in length, through which the unoccupied bronchus may obtain its air. The stock sizes and lengths as made by Fischer of Freiburg are: Adult, 9 mm. in diameter × 18, 25, 35, 41 cm. in length; children, 7 mm. in diameter × 13, 18, 23, 28, 35, 41 cm. in length. When the deeper parts of the bronchial tree were explored extension tubes of small diameter were passed down inside these tubes. The bronchoscope was introduced through the split-tube-spatula described above (p. 300) under ocular inspection. Although tubes with slanting orifices have certain advantages, Killian finds that foreign bodies are less easily drawn into them than into the straight-cut tubes. Killian now uses a bronchoscope devised by one of his assistants, Brüning; this consists of a tube-spatula made long enough to enter the trachea through the glottis, which it can be easily made to do by reason of a bevelled end, the lip of which can be introduced between the cords under visual control. Through the spatula

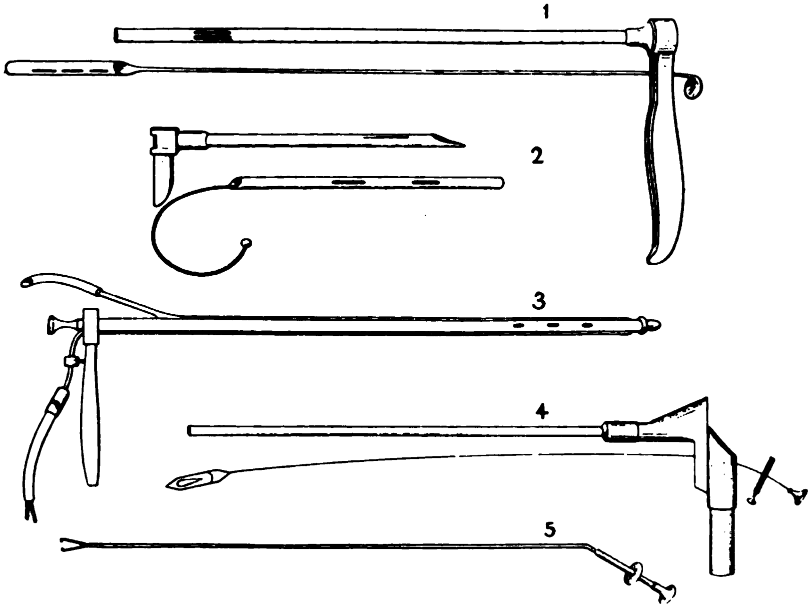

FIG. 39.—1. Killian's bronchoscope and extension tube; the oesophagoscope differs in dimensions and in the absence of fenestrae. 2. Brüning's combined tube-spatula and extension bronchoscope; used with electroscope (Fig. 37). 3. Jackson's bronchoscope with obturator and channels for suction and for the lamp-carrier (*vide* Fig. 35); the oesophagoscope and gastroscope differ from this merely in size. 4. Gottstein's bronchoscope with Casper's prism lamp fixed to funnel-shaped handle and mandril for opening glottis. 5. Killian's type of forceps.

is passed an extension tube capable at its full insertion of reaching a point 40 cm. distant. Thanks to the ingenious device of a watch-spring-like handle which fits in a groove in the tube-spatula, the lumen of the latter is not embarrassed by this complication. The tube-spatulas vary according to their diameter from 15 to 20 cm. in length, whilst the extension tube fully inserted doubles that length. Consequently a full-sized tube, capable of reaching well beyond the second branch of the right main bronchus, may be carried in an ordinary hand-bag. In order not to lose this advantage Brüning has devised a very ingenious telescoping forceps on the same watch-spring principle. Three sizes of these tubes are said to suffice for most cases. Illumination is obtained by Kirstein's lamp (Fig. 33), and for purposes of demonstration this or a Casper's prism lamp may be clipped on to the handle. Quite recently Brüning has devised a handle of the rectangular type, with a Kirstein's lamp fitted upon a slide, whereby it may be withdrawn some 3 or 4 inches away from the upper end of the bronchoscope. The best possible illumination results are obtained by delicate focussing and centring adjustments, and when the slide is raised, forceps and probes can be introduced without difficulty. The eye is protected from expectorations by a glass diaphragm, and arrangements are made for ready access to the mirror for cleansing purposes (*vide* Fig. 37). A terrestrial telescope may be added to obtain a magnified picture when desirable. Von Mikulicz and Gottstein employ the prism lamp, but with the addition that they have it permanently fixed in front of a fairly large funnel-shaped hand-piece, to which bronchoscope tubes of various sizes are readily adjusted. Gottstein does not find that the prism, which projects half across the line of vision, interferes with the manipulations of instruments. Both Killian and Gottstein have devised tubes with a suction apparatus inserted into the walls, and Jackson until quite recently had his tubes made on this plan. Von Schroetter has arranged a tube and illuminator in one; a crown of four small zirconium lamps is contained in a metal case forming the proximal end of the tube. The light impinges upon the upper edge of a glass tube which is carried right down to the bottom of the metal tube, forming as it were a lining to the latter. The light is remitted from the lower end of the glass tube in the manner well known in the cold lamps used in bladder surgery. Fletcher Ingals and Chevalier Jackson, following the example of Einhorn and Glücksmann with their oesophagoscopes, make use of a small lamp placed just within the distal end of the tube. In Jackson's instrument the wall of the tube is supplied with two channels, one for the light-carrier and one for a suction apparatus. The

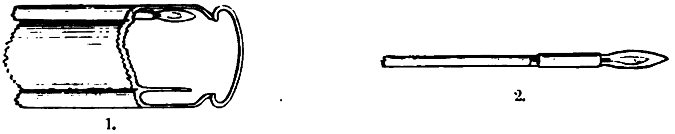
1. 2.

FIG. 40.—Jackson's bronchoscope. 1. Lower end shewing suction channel and lamp in position; 2. Lamp, actual size.

lumen is somewhat curtailed by these insets, but a good white light is obtained close to the objective, and in the case of long tubes the illumination is more brilliant than that obtained with Kirstein's head-lamp. The lamps run at a low voltage (*c.* 2 volts), do not become excessively hot, and do not crack when fluid comes in contact with them. It is said that no difficulty arises from their obscuration by blood and mucus, and that forceps are more easily manipulated with the light at the distal end of their shafts. I find in practice upon the phantom that it is easier to judge distances when the proximal illumination is employed, and while conscious of bias in favour of Killian's instrument, from the valuable instruction received from von Eicken, Prof. Killian's chief assistant, at Freiburg, I suggest that those who propose to commence the study of bronchoscopy should begin with Kirstein's lamp and the simpler tubes. With these there is no difficulty in obtaining a sufficiently brilliant illumination, particularly if the interior of the tube be kept polished, whilst distracting reflections from the side are avoided if a mat surface is given to the distal 2 or 3 inches, which then shew up as a dark frame around the mucous membrane picture. The more complex instruments are expensive, and are at present undergoing constant modifications.

Difficulty arises mainly from the presence of excessive secretion such as may occur in cases of old long-standing foreign bodies, in bronchiectasis, and other conditions. This can be obviated by repeated swabbing, and by the use of a small flat tube connected with a suction apparatus actuated by a hand-pump. The suction tube must not be so small as readily to become blocked by tenacious mucus and blood-clot. Besides wool-swab-holders the surgeon should be supplied with probes, some of them tip-tilted at the end, and with blunt and sharp hooks. These instruments must be preserved in a perfectly straight condition, with the exception of the proximal inch, which is bent at an angle to remove the fingers from the line of vision.

Gottstein uses a fenestrated wedge in order to separate the vocal cords, but an ordinary conical bougie rapidly inserted will serve this purpose, when, as seldom happens, it is necessary to do more than merely wait for an act of inspiration. A movable hook of the gall-stone pattern is valuable, and Jackson has devised a ring movable in the same manner for passing below an open safety-pin lying with its point upwards. The pin is closed by thrusting its hinge end into the ring by means of a small fork, a device originated by Mosher.

The forceps used are of the tube pattern, and it is essential that the jaws should not, in closing, be drawn into the tube. Killian's latest pattern of forceps with extremely delicate shafts is very satisfactory, and Brüning's telescopic development of them is admirable, and has the additional advantage of a lock-nut which secures a foreign body when once it is seized. Various forms of jaw must be obtained, the most useful being the ordinary mouse-tooth, cutting punch for new growths, von Eicken's with rectangular beak for seizing needles, and the bean-forceps fashioned like midwifery forceps. Morton's spear-hook is valuable for securing a flat object impacted so as to present as a diaphragm in the bronchial lumen, and Killian's expanding catch is useful in the removal of small hollow bodies.

At the suggestion of Dr. Wall I have had small funnel-shaped orifices made at the side of the proximal end of the bronchoscope through which the chloroform vapour may be pumped without interfering with the view.

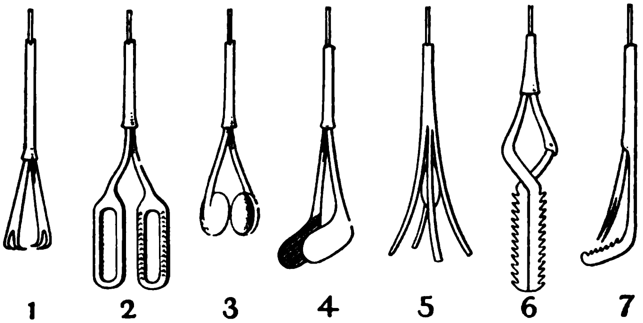

FIG. 41. Forceps ends. 1. Mouse-tooth ; 2. Bean-forceps ; 3. Cutting, for removal of specimens of new growths ; 4. von Eicken's for needles ; 5. Killian's spreading probe, for small hollow bodies ; 6. For hollow bodies ; 7. Morton's spear-hook for shirt-studs.

The operator will find that the vapour is extremely irritating to the conjunctiva, and it is advisable to wear glasses not only on this account but also to prevent infection from expectoration. Slightly + glasses are valuable in magnifying the bronchial picture.

Indications for Tracheoscopy and Bronchoscopy.—Though these endoscopic methods owe their present popularity to the numerous successful instances of removal of foreign bodies, their indications are by no means limited to such cases, for at Vienna and elsewhere they are frequently employed in the diagnosis and operative treatment of scleroma and hydatid cysts, as well as of bronchiectasis and chronic bronchitis. Cases of unexplained persistent cough have been cured by topical applications to tracheal and bronchial ulcers, usually syphilitic, whilst stenoses due either to syphilis or trauma have been dilated. Jackson describes a tent introducer for cases of stenosis, and von Schroetter has introduced aluminium dilators. Several cases of papilloma about the tracheal bifurcation in children have been dealt with, and also two cases of multiple growths of stony hardness. Diphtheritic membranes have been removed from the bronchi, and Pieniazek, in the early days, repeatedly removed dried suffocative secretions from tuberculous cases. Ozaena crusts have also been removed. The method is extremely valuable in cases of suffocative plunging goitre, by indicating which lobe should be removed ; and Jackson records a case of thymic asthma cured by operation after tracheoscopic diagnosis. Cases of dyspnoea associated with oesophageal and mediastinal growths may be diagnosed, and the pulsations of an aortic aneurysm, compressing the trachea, are easily seen. It is important not to confuse these pulsations with the impact received from the aorta by the left bronchus, the expansile character of the aneurysmal phenomenon when the cartilages have been absorbed giving a curious wave-like flapping appearance to the bulging tracheal wall.

These methods have undoubtedly gained their reputation from the excellent statistics with regard to the removal of foreign bodies by their means. Gottstein (16) gives detailed references to 135 cases up to the end of 1905; Killian, writing in June 1907, refers to no less than 159 cases, the results of which are definitely known, and probably 200 cases could now be collected. Of the 159 cases fully reported, 21 died (2 from cocaine poisoning, 2 from impaction, 1 from suffocation, 16 from pulmonary sequels (11 of these after removal of the foreign body)), 54 were completely successful by superior, and 63 by inferior, bronchoscopy. Killian's personal record is, however, even better; of 18 cases only 1 died, and that six months after the operation; in 1 the body was coughed up, and in 2 no foreign body was found. Of the successful cases, 12 were by superior bronchoscopy, and his colleagues von Eicken and Brüning have since added 2 successful cases by the superior method. It is impossible to compare these statistics with those of cases in which bronchoscopy has not been employed, because no complete statistics of the latter class are available. It may, however, be stated that removal by operation through the posterior mediastinum is almost always fatal, and it may be added that thoracic operations afford hope of success practically only in those cases in which the spikes of barley and other cereals make their way to the periphery of the lung, and give rise to a localised abscess close to the surface.

Killian in his record of 164 collected cases finds it important from a clinical point of view to divide the hard from the soft foreign bodies, and gives the following figures:—

Hard Foreign Bodies.—Needles, 12: cause little disturbance, and no danger is incurred by a brief delay; he advises von Eicken's angle-jawed forceps covered with thin rubber tubing. Nails, 6: 2 were removed by the electro-magnet. Nibs, 2. Denture, 1. Pieces of metal, 12: if smooth, use Brüning's locking forceps with rubber on jaws. Coins, 5: generally diagonally impacted. Pebbles, 4: smooth round bodies may be very firmly impacted in a bronchus which they fit exactly. Cubical bodies, 6. Buttons and beads, 3. Cherry stones, 2. Pills. Coffee beans, 4: all these to be removed with the bean-forceps fashioned like midwifery forceps. Apricot and prune stones, 4: rough and pointed. Shirt and collar studs, 7: if the base engages transversely to the lumen of a bronchus, pus may be expected behind it; Morton's spear-hook was designed for such a case in Killian's practice. Bones, 31: these scratch, become impacted, and cause severe lesions in the lungs; teeth, as they are infective, cause very severe pulmonary trouble. Hollow bodies, 16: for small whistles, pencil-holders, and the like, Killian's spreading forceps may be required.

Foreign Bodies, not Hard.—Wood, cork, nutshells, 9: care must be taken not to break these objects; a case of successful removal of a nutshell by superior bronchoscopy from the left bronchus has been reported by Dr. D. R.

Paterson (53). Seeds of melon, orange, and grapes, 6. Down, cotton, meat, quill, 5. Fruit leaves, etc., 4: I have removed from the trachea of a child an object exactly like a large nasal polypus, measuring about $1\frac{1}{2} \times 1 \times \frac{1}{3}$ of an inch; on drying, this proved to be a piece of maize leaf. Cereal spikes, 2: these wander; 25 per cent fatal. Expansile fruit-kernels, beans, etc., 18. (In more than 100 pre-bronchoscopic records referring to beans the mortality was 39 per cent.) Beans are very commonly met with; use bean-forceps and take care not to cause fracture; von Eicken had a case in which a bean broke into many pieces, and tracheotomy was required. These objects swell, cause complete blocking and consequent pneumonia. It is probably best to employ inferior bronchoscopy at the outset.

Technique of Superior Tracheoscopy and Bronchoscopy.—With regard to previous preparation of the patient with morphine or codeine, anaesthetisation and position, and the organisation of assistance, the reader is referred to the description given on p. 300. In tolerant patients I have found superior bronchoscopy perfectly feasible under cocaine and in the sitting posture, but, as previously stated, the composure of the patient and the control of the operator is greater when the lateral posture is adopted. Some surgeons prefer the supine decubitus, but the flexion of the neck certainly causes more distress to a conscious patient in this position, and the trickling of saliva into the posterior nares gives him great discomfort.[1]

When a foreign body is definitely located in the bronchus, the rule is to place the patient so that the side of lesion is uppermost, as this is said to facilitate the removal not only of the body but of the secretions. The head end of the table may be tilted downwards with some advantage. Since a long tube has to be introduced exactly in the tracheal axis, it should not, as in the case of the direct laryngoscopy, be introduced in the middle line but at the angle of the mouth; the angle opposite to that of the affected bronchus is chosen, and should be the lower one as the patient lies on the table. The first assistant (p. 301) must therefore not only extend the head after drawing it over the end of the table, but also rotate it and flex it upwards so as to bring the upper molar teeth of the lower side into the line of the tracheal axis. As a rule a third assistant should be present and concern himself solely with the suction apparatus, a spare cannula being ready in case of blocking by tenacious mucus. The second assistant will arrange his wool-swabs and instruments as described on p. 301.

The surgeon will select the broadest and shortest tube likely to be serviceable, being guided by the age and the sex of the patient, the distance from the 6th cervical to the interval between the 4th and 5th dorsal spines, and the skiagraphic data if available. The size of the foreign body does not greatly concern him in the choice of the tube, for if it proves too large to enter the lumen, both tube and object will be withdrawn together. In any case all preparations necessary for tracheotomy should be made, in case the body should become impacted in the subglottis, and the surgeon should clearly indicate to the second assistant the names of the various forceps and tubes, so to avoid confusion in an emergency. After cocainisation of the larynx he introduces the tube-spatula, and then passes the bronchoscope carefully through the glottis under inspection, waiting for an inspiration to do so, or introducing a taper-bougie if spasm occur. He next applies cocaine to the bifurcation, and, after an interval, to the affected bronchus. The parts about a foreign body are always highly sensitive and require a good deal of the drug. The complete abolition of the cough reflex is an end rather to be hoped for than attained.

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[1] *Vide* page 319 for code of signals between patient and surgeon.

Meanwhile the tube-spatula has been separated into its two halves and removed, and the bronchoscope, held in the left hand and rotated so that its lateral fenestrations are directed towards the orifice of the sound bronchus, is passed down to the seat of disease. The tube should be kept from contact with the teeth with a piece of rubber sheet, and great care should be taken to see that the lips are not pinched. A gag is generally desirable. The tube should be vaselined externally before introduction. When Brüning's telescopic apparatus is used, the tube-spatula, which is long enough to enter the trachea, remains as part of the bronchoscope; the lower, movable section of the instrument has two pairs of lateral fenestrations to suit different depths of introduction. The tube must be passed down carefully and slowly, note being taken of the centimetre marks on its outer surface. The bifurcation is soon reached, and when the patient is lying on the right side, the left bronchus is often entered before the operator is aware of this, and its apparently horizontal lower surface may be mistaken for an abnormal structure. The bronchus straightens out as the tube slips down it. It is most essential to recognise the triangular ridge between a dorsal and ventral branch as such, and not to mistake it for a foreign body. Under general anaesthesia a considerable time may be occupied in examining the parts without doing any harm. After very prolonged retention of the tube, two or three cases have been reported of oedema of the larynx necessitating tracheotomy, but I have on two occasions (once in an infant) kept a tube in for nearly an hour with no worse result than slight hoarseness of a day's duration, though both patients had foul and profuse bronchorrhoea, which caused the difficulty and delay. In such cases the pump must be frequently passed, in addition to the swabbing. I have no experience of the use of Jackson's tube with the inset suction pipe in a bronchorrhoeic case. The remarks on swab-holders on p. 301 also apply here.

With regard to the manipulation of probes and forceps it may be said that the difficulty in judging distances and excluding the disconcerting sensations which arise from the necessary contact with the metal tube can only be overcome by frequent practice upon a rubber phantom, such as that devised by Killian, or a set of branching rubber tubes, which serve much the same purpose. When the nature of the foreign body is known, actual practice should be made with its duplicate placed in the phantom. The remarks appended to the list of foreign bodies on p. 311 will assist in the selection of suitable forceps. It must be remembered that if a body is partially withdrawn and allowed to drop into the healthy bronchus, severe consequences may speedily ensue. It can never be safe to drag forcibly upon a rough or possibly barbed body. In the case of the oesophagus, Killian had on one occasion to cut a denture into three parts with a cautery snare before effecting its removal. In long-standing cases, granulations and cicatricial stenosis may add great difficulty to the operation. In view of the possibility of laryngeal oedema and pulmonary sequels, no case should be sent out of hospital for at least two days. These dangers, though no doubt real enough, are infinitely less than those which accompany the expectant attitude, which in many cases will inevitably lead to a septic condition of the lung, and not infrequently to suffocation by shifting of the foreign body and subsequent impaction either in the glottis or in the other bronchus. It is true that foreign bodies have been tolerated for many years by patients in comparatively good health; thus, in a case known to me a piece of glass was coughed up after ten years' retention. The history given by this patient was persistently regarded as a phantom of her brain, and in two cases a similar history has been disregarded, and a foreign body has been found after death. Where such a history is given, and the physical signs and symptoms in the least degree corroborate it, a bronchoscopic examination should be made, even though *x*-rays give a negative result.

DIRECT OESOPHAGOSCOPY.—The history of oesophagoscopy goes back much further than that of bronchoscopy, and may be studied in the works of Starck (70) and Gottstein (17). Though various indirect or reflecting apparatus have been used, notably that of Mackenzie, Kussmaul in 1868 was the first to realise a direct oesophagoscopy by passing a straight rigid steel tube into the person of a professional sword swallower at Freiburg.

Anatomy.—The walls of the oesophagus are so distensible that the statement that the diameter of the lumen is an inch in the lateral and ¾ of an inch in the sagittal direction is of no great practical interest. At the upper and lower ends, however, it is reduced to a slit about an inch in length. The direction of this slit is, of course, transverse behind the cricoid, whilst the orifice in the diaphragm runs obliquely from behind forwards to the left. As to the longitudinal measurements, Starck gives the following figures, which are of practical importance:—

	Teeth to Cricoid.	To Bifurcation of Trachea.	To Cardia.	Length of Oesophagus.
1 year .	10 cm. (4 in.)	14 cm. (5½ in.)	22 cm. (8¾ in.)	12 cm. (4¾ in.)
10 years .	10 cm. (4 in.)	18 cm. (7 in.)	28 cm. (11 in.)	18 cm. (7 in.)
Adult .	15 cm. (6 in.)	26 cm. (10¼ in.)	40 cm. (15¾ in.)	25 cm. (10 in.)

As slight constrictions occur behind the left bronchus and where the aorta arches over the tube, the following data (Starck) may be useful in estimating distances in a given case:—The cricoid constriction is opposite the 6th cervical vertebra; the aortic constriction is opposite the 4th dorsal, the constriction behind the left bronchus is opposite the 6th, and the diaphragmatic constriction is opposite the 10th dorsal vertebra.

Instruments.—The statements with regard to illumination and to tubes made above (p. 299) apply to oesophagoscopy, with the exception that the tubes are both longer and wider. Killian uses tubes with a diameter as large as 13 mm., and it is advisable to purchase two of this calibre, one 52 and one 26 cm. in length. For children the adult bronchoscopes will suffice perfectly well; a sleeve must, however, be slipped over the fenestrations, for the mucous membrane which bulges through them obscures the view and runs a risk of laceration. The illumination at the lower end of a 52 cm. tube with polished sides is surprisingly good with a Kirstein's lamp. Jackson employs a tube with a terminal lamp, and states that with very long tubes used for gastroscopy, for example 80 cm., a very much whiter and better illumination is obtained. With this I quite agree, but I have not yet made use in the stomach of Brüning's apparatus, which is stated to give a brilliant illumination. Jackson's instruments are furnished with two inset lateral channels, one for the introduction of the lamp and its carrier, one for suction purposes. The same swab-holders, probs, hooks, and forceps will be used as in the case of bronchoscopy, and it is desirable in making purchases to see that the accessory instruments are long enough to serve the two purposes. Brüning's telescopic forceps are particularly useful as lending themselves to easy manipulation in dealing with the upper end of the organ. Round-ended flexible bougies must be provided. They must be long enough to project an inch beyond the tube orifice, which they must closely fit to prevent wounding of the mucous membrane during the passage of the cricoid constriction. Taper-ended bougies are apt to catch in the anterior lip of the pyriform fossa and to buckle up, and should not be used in introducing the tube, though they may, of course, be called for in the treatment of strictures through the oesophagoscope.

The indications and contra-indications for endoscopy are the same as those which controlled the passage of the bougie before the oesophagoscope came into use. Now, as then, cases of large aneurysms, mediastinal tumours, or foreign bodies accompanied by surgical emphysema and fever, should not be complicated by the passage of instruments. With these exceptions all the conditions accompanied with pain or difficulty of swallowing can safely be examined by this method undertaken with cautious and delicate manipulation under the control of vision. Malignant disease can be diagnosed with certainty by the removal of small fragments for microscopic examination, and the tortuous lumen of malignant strictures can be safely threaded with a rubber feeding-tube passed under the guidance of the eye. A considerable number of cicatricial strictures, both traumatic and syphilitic, have been successfully dilated. Benign growths have been found to exist much more frequently than was formerly supposed, and both these and a small sarcoma (Killian) have been removed with success. The diagnosis of stenosis from extrinsic pressure caused by enlarged glands and mediastinal growths has been made.

The method has a special value in cardiospasm and dilatation, for these associated conditions have frequently been found to be due to the presence of unsuspected ulcerations, and have readily yielded to the application of silver nitrate to the diseased part. In these cases of cardiospasm Jackson has definitely located a spastic condition in the short section of the gullet between the diaphragmatic hiatus and the cardia, with dilatation above it, and in two instances a peptic ulcer of the abdominal oesophagus was found. He differentiates a phrenospasm, due to a spastic condition of the diaphragm with superjacent dilatation, the phenomenon disappearing under general anaesthesia. Oesophagospasm, a general spastic condition of the entire tube, is the common condition in globus hystericus; this also disappears under general anaesthesia. With regard to stricture at the lower end of the oesophagus, it may in general be said that malignant disease seldom presents a difficulty to the passage of the bougie passed under inspection, and that therefore the existence of such a difficulty is a favourable diagnostic sign. It is not uncommon to see hysterical women with partial anaesthesia of the pharynx and defective teeth in whom the symptoms simulate organic stricture. The passage of the oesophagoscope is easy in such cases and conclusive in its result to the mind of the patient as well as to the surgeon, who will probably effect a cure by ordering a set of false teeth. In cases of oesophagospasm, when the absence of any organic lesion has been definitely determined by inspection, the repeated passage of a bougie may be safely entrusted to a nurse or the patient herself.

In certain cases of dilated oesophagus organic lesions have unexpectedly been discovered. Pouches (*vide* Vol. III. p. 332) fall into two main categories—(1) Traction pouches due to cicatricial contraction from without; (2) pulsion or pressure pouches resembling aneurysms, and due to the gradual dilatation of a circumscribed weak portion of the wall subjected to internal pressure. Autoscopy of these pulsion pouches presents some difficulty; they occur on the posterior aspect of the tube, just above the cricoid constriction, and when full of food cause difficulty in swallowing, by compressing the oesophagus from behind. When the oesophagoscope is used it passes readily into the pouch; consequently the true orifice of the gullet, which takes the form of a narrow slit just below the edge of the cricoid, is very easily missed. Unusual facility in the passage of the instrument past the cricoid should warn the surgeon of the possibility of this condition. I have examined one such case in which a star-shaped scar at the bottom of a pouch was highly suggestive of a tight cicatricial stricture with a superjacent dilatation, a circumstance which at first led to an erroneous diagnosis.

With regard to foreign bodies the general statement may be made that when surgical emphysema and fever are present, oesophagotomy is indicated, and that in all other cases in which pain or difficulty in swallowing suggests, or the *x*-rays prove their presence in the gullet, the oesophagoscope should be used. The blind introduction of the probang and coin-catcher has beyond any question been responsible for many deaths. I have seen fatal mediastinitis result from the passage of a bougie in a case in which such an apparently harmless foreign body as a coin had caused ulceration. The coin-catcher is a dangerous instrument, and has been known to tear an oesophagus with fatal results. Though the oesophagoscope may not be successful in the removal of a large and jagged foreign body, and in such a case oesophagotomy is generally necessary, its use does not involve any additional danger, the damaged area being approached under ocular inspection. In a case with severe dysphagia and progressive oedema about the arytaenoids and cricoid cartilages following ingestion of a sharp bone, I found a wound of the lateral wall, and under chloroform removed some twenty fragments, which proved to be pieces of meat retained in the inflamed and paretic oesophagus, the operation occupying nearly half an hour. In spite of these prolonged manipulations the patient was perfectly well after a day in bed with sterile iced liquid food. In a case of von Eicken's, which I watched, a sharp bone had been thrust down by the previous use of the bougie, with consequent impaction in the cardia, laceration, and some fever. It was carefully passed on, under oesophagoscopic inspection, into the stomach with a favourable issue. These are merely examples illustrating the statement that whereas the blind use of the bougie may lead to disastrous results, the endoscope may with impunity be manipulated in a wounded gullet, if proper care be taken. I can recall one instance only, and that in the early days of the method, in which temporary exacerbation of symptoms followed the introduction of the tube; this was in an alcoholic patient with extensive syphilitic ulceration of the hypopharynx.

Technique.—Oesophagoscopy should, as a matter of routine, be preceded by inspection of the cricoid region with the mirror. If no information be obtained, the posterior wall of the cricoid and upper end of the gullet should be examined by the method of hypopharyngoscopy devised by von Eicken. This consists in drawing the larynx away from the spine by means of a long and strong laryngeal probe, its tip covered with a rubber tube, which is inserted into the anterior commissure after thorough cocainisation. By this means the evidence of a pouch or of malignant disease at the upper end may be detected with the laryngeal mirror employed in the usual manner. If this be unsuccessful, the tube-spatula should be inserted as described under laryngoscopy, but with its lip behind the arytaenoids.

Oesophagoscopy is easily tolerated under cocaine by the patient who sits upon a low stool. For a simple inspection assistance may be dispensed with, but in difficult or complicated cases a first and second assistant are necessary, the first to support the back and hold the head in the correct position, the second to manage the wool-swabs and instruments, which must be handed to the surgeon in the position in which they will be inserted, and taken from him when withdrawn. With chloroform, and in many cases with cocaine anaesthesia, either the supine or right lateral position can be used, and then the office of the first assistant in securely holding the head (p. 301) is of the utmost importance. In cases of dilatation, a third assistant will be necessary to look after the suction pump, which readily becomes choked with food particles. As the oesophagus passes downwards and to the left, the tube is introduced through the right angle of the mouth. With general anaesthesia a gag will be necessary, and should be held by the first assistant with his left hand passed under the neck, while he supports the head with his right hand and knee. Care must be taken to protect the lips from being pinched between the tube and the teeth, the latter being protected with sheet rubber. Pressure upon the molar teeth is the only incident which causes real distress to the majority of conscious patients. As in bronchoscopy, a small dose of morphine may be given an hour beforehand, and there should be an interval of four hours since the last meal. In oesophagoscopy all instruments must be warmed by immersion in a tall can of hot sterile water; for if this is not done a column of mist is produced as soon as the first mop used is withdrawn, and there being no respiratory current, as in bronchoscopy, to disperse it, the view is effectually obscured.

In the case of an adult, a short large tube, 26 cm. by 13 mm., is warmed and thoroughly vaselined without, and a flexible round-ended bougie, exactly fitting it, is introduced to project an inch beyond the extremity. The theatre is darkened, and Kirstein's head-lamp properly adjusted as close as possible to the right eye. In the case of a sitting patient the surgeon stands facing him, and holding the instrument in the right hand he introduces his left forefinger into the right pyriform fossa, and with it guides the tip of the bougie into that channel. The patient is requested to swallow, and the bougie and tube are gently passed down through the cricoid constriction. Some assistance may be given by forward traction on the hyoid with the left forefinger. If definite resistance be encountered, the bougie is removed, the tube slipped down to the obstruction, and ocular inspection made, the surgeon bending over the patient to do this. When the cricoid constriction has been passed, no further difficulty exists in a normal gullet, the tube slipping easily down, the picture being constantly watched both during the insertion and the subsequent slow withdrawal. As the patient usually complains of the prolonged retention of the tube rather than of its introduction, it is well to withdraw the short tube if nothing is found, and, after an interval, to insert the long tube precisely in the same manner.[1]

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[1] I have recently had several opportunities of using Brüning's oesophagoscope, constructed in the same manner as his bronchoscope; and I find it simpler to introduce and both safer and more efficient, as the cricoid constriction is passed under ocular inspection.

With a patient in the prone position the surgeon stands at the head and inserts the left forefinger with his arm extended, and passes the bougie and tube into the right pyriform fossa. Once the tube has been inserted, the surgeon should sit down for the further manipulations. The first assistant must take great care that rotation of the head does not permit pressure from the teeth to displace the lower end of the instrument.

It may be said of oesophagoscopy that it is a perfectly simple manoeuvre, the chief difficulty arising from fluid contents when present. If the surgeon has practised throwing the light and judging distances upon the dummy, he will find little difficulty in the passage of dilating bougies, probing, swabbing ulcers, and removing portions of new growths. Respiration is not interfered with, but the position is one of uncomfortable restraint. An honourable compact should be made with the patient, so that he may know that the tube will be immediately withdrawn when he makes a pre-arranged signal with his hands, such as the double tap employed in Ju-Jitsu, whilst a single tap may be used to signify that the lips or teeth are the source of pain.

It is, of course, useless to proceed with a conscious patient who loses courage at the critical moment, that is to say, at the passage of the cricoid constriction; but, apart from such instances, I have only once found the method impracticable, and then in the case of an old man with considerable stiffening of the dorsal and cervical regions of the spine. In well-marked lordosis the method is, of course, contraindicated. This facility must not, however, engender the rash and careless employment of the method. Fatalities have been reported from perforation at the seat of malignant disease, and in aneurysm the risks are obvious. I was recently asked to report on a case of dysphagia, but from some accident was unable to undertake the examination. Twelve hours later the patient died suddenly from perforation of the aorta by a syphilitic ulcer. This event would undoubtedly have been put down to such an examination, had it been made.

DIRECT GASTROSCOPY.—Instruments resembling the vesical endoscope have in the past been employed for the examination of the stomach, but Chevalier Jackson (31) has now proved that a very complete direct examination of the organ may be made with an oesophagoscope elongated to 70 or 80 centimetres. His gastroscope is, like his other instruments, illuminated by a small lamp at the distal end, and his principle, unlike that of previous workers, is to examine the organ in a perfectly empty condition, going carefully over the whole area as the walls fall collapsed against the end of the tube. Not only must the organ be in a fasting condition, but all fluid must be removed; this object is attained by the use of a suction cannula let into the walls of the tube. If deep ether narcosis is employed, relaxation of the diaphragm is obtained, and it is found that the orifice of the tube can be moved a considerable distance from right to left and from front to back, the instrument pivoting about an axis situated fairly high up in the thorax. Jackson has found no difficulty in making the tube point in turn towards the right and left anterior superior iliac spines, and he shews skiagrams with the tube in the right and left iliac fossae. The tip of the tube can be made to describe an ellipse measuring 5 by 15 cm., and this freedom, coupled with manipulation from without, gives sufficient range for the examination of the whole surface in favourable cases.

The technique is practically the same as that for oesophagoscopy under anaesthesia, the tube being inserted through the right angle of the mouth in the supine posture. So far, Jackson has not found any evidence of dangers other than those which contra-indicate oesophagoscopy and general anaesthesia.

The colour of the gastric mucous membrane, which is deeply folded, varies greatly even in the same individual, and, when injected, contrasts markedly with the paler oesophagus. The brilliant white illumination from a lamp at the distal end of the tube is stated to be an essential element in gastroscopy by Jackson, who has examined cases of peptic ulcer and of malignant disease, and who gives drawings of several normal and pathological cases.

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E. W.

FOREIGN BODIES IN THE AIR- AND UPPER FOOD-PASSAGES

By Sir Felix Semon, K.C.V.O., M.D., F.R.C.P., and P. Watson Williams, M.D.

The subject of foreign bodies in the fauces, pharynx, larynx, and trachea may be conveniently considered in one article for two reasons; first, on account of the very important fact that the power of localisation of sensations felt in the throat is extremely defective, and sensations arising in any part of this region are generally subjectively referred to one common region, namely, to the front part of the neck corresponding to the larynx and upper part of the trachea, the laryngo-tracheal region; and, secondly, because the invading body is obviously liable at any moment to pass from one region to another, with or without modification in the symptoms presented. These remarks do not apply to the same extent to the nasal passages proper (*vide* p. 57).

It is unnecessary to enumerate the various foreign bodies that may become impacted in the throat; our purpose is mainly to consider the chief difficulties that may be encountered in the diagnosis, and the principles that should guide us in the treatment of these cases.

In a considerable proportion of the patients who present themselves for the removal of foreign bodies in the throat, the foreign body has already been dislodged, and it is the persistent sensation only which makes the patient believe that it is still there. These after-sensations of pain, pricking, and soreness, or of the actual presence of the foreign body, are apparently far more lasting in this region than in other sensitive parts, such as the eye; and it is important to remember that in spite of the most positive assurances, even of educated persons, that the foreign body is still present, it may long have passed down, leaving behind, however, strangely vivid and persistent after-sensations: more especially is this the case when foreign bodies have been impacted in the pharynx, tonsils, and the upper part of the oesophagus. On the other hand, we cannot too strongly insist on the necessity for a most thorough and methodical examination of all the parts in question; and only after a positive exclusion of the possibility of the continued presence of the foreign body are we warranted in arriving at a diagnosis of a persistent after-sensation only, and in telling the patient that the foreign body is no longer impacted.

In all cases except those of immediate urgency (see p. 326 under the head of "Treatment") the examination should be begun by inspection, and not by digital exploration. Palpation may be desirable or necessary when inspection has failed; but it is always attended with the risk of dislodging the foreign body and driving it farther down, possibly into the lower air-passages; whilst in the case of small pointed bodies, such as fine fish-bones, pins, and needles, which are already deeply buried in the tissues, the portion still projecting may be pushed in even farther and completely buried, whereby subsequent attempts at removal are made more difficult if not altogether frustrated. For the same reason, if drugs, such as cocaine, eucaine, novocaine, alypin, are used to diminish the soreness and irritability of the parts, they should be applied by means of a spray, and not by a brush, which is open to the risk of producing the same undesirable result as digital exploration. For the inspection of the throat a good light is essential, and the examination should extend to every region in turn, and not be limited to an inspection of the one part which the patient indicates as being in his opinion the place where the foreign body is lodged, inasmuch as the subjective sensation of localisation is very deceptive. This is well illustrated by the personal experience of one of us (F. S.), in whom the sensations caused by a piece of partridge bone impacted in the throat were felt at a distance of at least two or three inches from the spot where it was really impacted. A patient may positively state, as he did in this instance, that he felt sure the foreign body was impacted in the region of the larynx, whereas in reality it had stuck in the posterior wall of the pharynx behind the uvula. This instance well exemplifies the necessity for a really methodical examination; and an observer who trusts the patient's statements in these cases, and only examines the laryngeal region, neglecting a thorough inspection of the fauces and naso-pharynx, may have the mortification of hearing afterwards that another physician had actually removed the foreign body from a part unsuspected.

It is advisable, therefore, to begin the examination with inspection of the fauces, particularly noting that the foreign body is not lying concealed by the anterior pillars of the fauces or by the tonsils. Next we should observe the glosso-epiglottic fossae and lingual tonsils, before thoroughly examining every part of the larynx and the upper end of the oesophagus with the largyngoscope; finally, the rhino-pharynx and hypo-pharynx should be explored. Particular care should be taken when the foreign body is a fish-bone; for, if so deeply impacted in the tissues that only a small part projects, it is sometimes extremely difficult to discover it; the more so as strings of tenacious saliva extending from one part of the throat to another often closely simulate it. In such cases examination with the probe, under the guidance of a good light and, if necessary, of the laryngoscope, ought to establish the actual existence of the supposed body in the tissues before an attempt is made to introduce forceps or other instruments for removal. Our means of detecting the presence of such foreign bodies as are impenetrable to *x*-rays in the upper air- and food-passages have been greatly enriched by the introduction of that method of examination. It has proved of the highest value in cases in which coins, buttons, needles, bones, and similar foreign bodies impenetrable to light have become impacted in these parts, and cannot be discovered by the ordinary methods. Equally invaluable for the detection as well as for the removal of foreign bodies in the lower pharynx, and particularly in the trachea and bronchi, are the methods of direct laryngoscopy introduced by Kirstein and by Killian, and von Eicken's method of hypopharyngoscopy (see p. 317).

It is impossible to form any definite classification of the foreign bodies that may be encountered in the different regions; but speaking generally, it may be said, as a rule, that only sharp-pointed bodies—such as pins, needles, and small-pointed pieces of meat or game-bones and fish-bones—become fixed in the fauces and rhino-pharynx, though they are equally apt to be caught in the larynx or oesophagus. Coins and small rounded bodies usually pass down till they are impacted in the larynx, oesophagus, or lower air-passages. In the larynx they are most apt to lodge in the pyriform sinuses, or to lie across the glottic opening, upon the ventricular bands, or between the ventricles of Morgagni.

Owing to the funnel-shaped narrowing of the lower end of the pharynx, and to the fact that the narrowest part is at the level of the cricoid cartilage, foreign bodies, if arrested on their passage downwards, are particularly apt to lodge at this spot. Bodies which pass into the trachea most frequently lie on the bifurcation, or pass into the right bronchus; the right bronchus being the seat of lodgment about twice as frequently as the left (*vide* p. 305). But sharp-pointed bodies are sometimes arrested in the trachea.

Symptoms.—Whilst the primary symptoms of a foreign body in the throat, and particularly in the larynx, are generally sufficiently obvious when the patient states the cause of his suffering, it is important to bear in mind that some cases of apparently sudden loss of consciousness may be due to occlusion of the glottis by a foreign body; and if summoned to a patient said to have had a fit or to have become suddenly unconscious while eating, the possibility of such an accident should not be forgotten.

In children especially, foreign bodies are liable to be swallowed or drawn into the air-passages unconsciously, where they may set up more or less acute dyspnoea or obstruction to deglutition. One of us (W. W.) has seen a case which presented all the symptoms of croupous laryngitis, and, dyspnoea becoming urgent, tracheotomy had been performed. The symptoms abruptly subsided when a piece of nutshell was coughed up and revealed the true character of the complaint, after it had been arrested for six days. This case aptly illustrates the necessity of thinking of foreign bodies when an obscure inflammatory affection or a swelling is seen in the air- or food-passages, even though no history of the impaction is obtainable. These remarks apply with no less force to cases of adult patients. Obstinate violent cough, followed after some time by expectoration of muco-purulent or purulent, sometimes blood-stained, and not rarely fetid secretion, should remind the observer of the possibility that these symptoms may be due to impaction of a foreign body in the air-passages, even though there is no history of such an accident, and should induce him to employ all the diagnostic helps enumerated above.

Haemorrhage may result from direct injury by a sharp body; thus, Rivington records a case in which a fish-bone, lodged in the pharynx, penetrated the common carotid and necessitated ligature of the artery. The puncture was believed to be due to the use of a probang whereby the fish-bone was pushed through the wall of the pharynx.

If a foreign body has lodged between the vocal cords, and, owing to the small size or peculiar position or shape of the invading body, acute asphyxia is not induced, aphonia may be the most notable symptom; and when it passes into the trachea or a bronchus, violent coughing and dyspnoea will be experienced. On the other hand, especially after the initial symptoms have passed off—such as pain, or coughing and dyspnoea, if the body lodge in the larynx, or the sensation of the presence of a foreign body if it lodge elsewhere,—there may be no indication whatever of its presence; nevertheless a careful exploration of the whole region should be made, as secondary mischief may subsequently arise.

Very frequently foreign bodies, which shortly after their impaction cause slight symptoms or none at all, may later be the source of most serious troubles. Secondary symptoms are generally of the nature of inflammation or ulceration, in consequence of which an abscess may form and the pus may burrow in the structures, or even set up suppuration in the mediastinum. In the larynx a foreign body, after originally giving but little trouble, may cause subsequent perichondritis and lasting disablement of the organ; or, after having remained in the larynx for some time, may become dislodged and fall into the lower air-passages, setting up most serious disease there. If a bronchus have been invaded, secondary pneumonia and pulmonary abscess or bronchiectasis are apt to supervene. Sometimes penetration of the structures in the neck causes extensive subcutaneous emphysema. In the oesophagus after a while it may either perforate the wall or lead to the formation of a pouch. Copper coins may give rise to metallic poisoning; and foreign bodies more or less occluding the oesophagus may produce such a degree of dysphagia as seriously to interfere with deglutition and nutrition.

All these contingencies are so grave that we cannot contemplate the impaction of a foreign body in these parts with indifference, even if at first it be unattended by serious symptoms.

Treatment.—We have already insisted that methodical investigation should precede any attempt at treatment in ordinary cases. When, however, the lodgment of a foreign body in the air-passages results in dyspnoea so urgent as to threaten immediate asphyxia, or has actually caused loss of consciousness, there is obviously no time for a careful examination of the throat; the forefinger should at once be cautiously passed down to the larynx, and, if the cause of the obstruction can be felt, the body may be dislodged; but it is important to avoid pushing the foreign body into the trachea. If nothing can be felt in the larynx, and the urgency of the case permit, the patient should be inverted. In this position a sharp blow on the back may dislodge the foreign body from the trachea or bronchus, and cause it to fall into the larynx, whence, possibly by inducing coughing, it may be expelled without tracheotomy. But if these efforts prove futile, tracheotomy should be performed promptly; and if, nevertheless, dyspnoea be still urgent the patient should again be inverted, and every effort made to cause the foreign body to pass into the upper region of the trachea, whence it may be extracted through the tracheotomy wound.

In the vast majority of patients who seek medical aid complaining of a foreign body in the larynx, the symptoms are less urgent; or, if at first alarming, the more acute manifestations of the presence of the impacted body have subsided; in these circumstances both the examination and the treatment can be carefully and methodically conducted.

Two principles should guide the practitioner in treating these cases:—First, no foreign body, the presence of which has actually been detected, should be permitted to remain impacted, even although at the time it may not produce any active symptoms; we have already pointed out the very serious secondary symptoms which may arise nevertheless. In the face of these risks it is hardly necessary to emphasise the importance of leaving no justifiable means of removing the foreign body untried. Secondly, no attempt should ever be made forcibly to ram down an angular or pointed foreign body. The danger of passing bougies or probangs for this purpose is self-evident; yet this risk is very frequently ignored, and consequently perforation of the carotid or the descending aorta, tearing or perforation of the pharyngeal and oesophageal walls, and many other serious results, have actually occurred.

Needless to say, no definite rules can be laid down as to the best method of removing the various foreign bodies that may become impacted in the regions in question; the practitioner must be guided in each case by (*a*) the nature and size of the foreign body, and (*b*) the spot in which it has become lodged. In cases of impaction of foreign bodies in the pharynx and rhino-pharyngeal cavity, forceps with indented blades will in most instances be the most suitable instrument, the curve of the forceps being adapted to the locality of the impaction.

When the body has passed down into the larynx, or into the lower air-passages, and when its form is rounded or circular, as of coins, beans, peas, and so forth, it is always worth while, before any instrumental interference, to try inversion and forcible shaking of the patient; the plan may even be adopted when the foreign body is pointed or angular. In a most remarkable case, seen by one of us (F. S.) and described by Mr. Pitts, an ear-ring, which had first become impacted in the larynx just below the vocal cord, and a few days afterwards had fallen into the left bronchus, was spontaneously evacuated by coughing about an hour after inversion and shaking had been tried, apparently without success.

Should the foreign body be fixed in the larynx itself, and should its nature be such as to allow of the hope of removing it by intra-laryngeal operation without injury, this plan of treatment will, of course, be preferable to an external incision. Should it be too large, however, or too irregular to justify such attempts, and should it, moreover, cause dyspnoea, tracheotomy might first be performed, and an attempt be made to get hold of it through the tracheotomy wound, or to dislodge it from the larynx into the pharynx, where it can, of course, be grasped more easily; or, if this should fail, tracheotomy may be carried forward to thyrotomy and the foreign body thus removed.

A similar plan of treatment is called for when foreign bodies of large size and angular shape have lodged in the trachea; and in cases in which the foreign body is situated in one of the bronchi, tracheotomy, followed by an attempt at extraction by means of very long slender forceps, is advisable. If the foreign body cannot be extracted at the time of the operation itself, it will be desirable not to insert a tracheotomy tube, but to keep the tracheal wound open by stitches in the trachea attached to an elastic band carried from the two sides of the wound round the neck posteriorly; thus, in the event of the foreign body becoming subsequently dislodged, it can easily be expectorated through the open wound during the act of coughing. The removal of bodies impacted in the oesophagus is dealt with on p. 317. Only when it is quite certain that the offending substance is of a soft or rounded form can it be justifiable to push it down into the stomach.

Many cases of foreign body impacted in the larynx, trachea, bronchi, or oesophagus, in which the difficulties in the way of locating and of removing them through the natural passages were insuperable, may now be successfully dealt with by the direct method of Killian, which is described at length in a separate section (see p. 299). Dr. Paterson in this country, and H. von Schroetter of Vienna, as well as many others, have been singularly successful in removing foreign bodies by Killian's method, whilst the inventor has removed a very large number (*vide* p. 311).

Skiagraphy has very frequently been used to locate the foreign body, and should never be omitted when its site is doubtful, provided, of course, the body is of such a nature that *x*-rays can be expected to prove serviceable. Of course extraordinary cases require special measures; the necessity of oesophagotomy or gastrotomy may even arise: but the problems of dealing with these various cases and the mode of treatment to be adopted are of a purely surgical kind and beyond the scope of the present article.

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F. S.
P. W. W.

DISEASES OF THE TRACHEA

By Sir FELIX SEMON, K.C.V.O., M.D., and P. WATSON WILLIAMS, M.D.

General Remarks.—As the trachea is merely an air-conducting tube without any other special functions, its diseases are, of course, subordinate in importance to those of the larynx on the one hand, and to those of the bronchi on the other. They would indeed hardly demand any special consideration, were it not that interference with the lumen of the tube may and does lead to grave general disturbances; and as the changes, which may bring about such interference, are of an extremely manifold character, a somewhat more detailed account of stenosis of the trachea, whether due to internal obstruction or to compression from without, is required, whilst its other diseases will be more summarily disposed of.

Tracheoscopy.—For full inspection of the lumen of the trachea and bronchi, the method of direct tracheoscopy and bronchoscopy, which is described fully on page 299, must be adopted; but much useful information may be gathered from the examination of the trachea by the simpler means of the laryngoscopic mirror, which should form a routine part of the examination of the larynx. For this purpose a brilliant light is even more essential than in laryngoscopy, as the illuminating rays must penetrate down the lumen of the trachea through the relatively narrow glottic chink during respiration. The patient should sit erect, without tilting up his chin, and the mouth should be slightly inclined forwards, on a somewhat higher level than the examiner's eye. This allows the laryngoscopic mirror to be held more horizontally than in ordinary laryngoscopy, the reflection in the mirror thus being thrown more directly down the trachea to the bifurcation. In cases in which tracheotomy has been performed, useful information may sometimes be obtained by the introduction of a very small mirror, the reflecting surface of which may, of course, be directed either upwards or downwards, into the trachea itself, and by illuminating the reflecting surface as in ordinary laryngoscopy.

When the bifurcation has come into view, the posterior tracheal wall can be followed upwards for some distance by gradually moving the mirror more horizontally, and the anterior wall by reversing the mirror. Similarly the lateral walls are brought into view by lateral tilting of the mirror. The upper portion of the normal trachea, however, is concealed by the narrowing of the air-passage immediately below the glottis. It should be noted that even normally the tracheal axis may not be quite vertical, and that pulsation can often be observed low down on the left side, presumably communicated from the pulmonary artery. The whitish-yellow rings of the trachea may be plainly seen, and under normal conditions there should be no lateral bulging of the tracheal walls. Not rarely distinct pulsation is visible at the region of the bifurcation, which is communicated to the trachea from the aorta.

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For clinical purposes the **Diseases of the Trachea** may be dealt with under the following headings :—

1. Defects and Malformations.
2. Injuries.
3. Inflammations (catarrhal, infective, perichondritis).
4. Syphilis.
5. Tuberculosis.
6. New growths (benign and malignant).
7. Stenosis from (*a*) internal obstruction, including foreign bodies; (*b*) from external compression.
8. Neuroses.

1. **Defects and Malformations.**—Congenital fistulas of the trachea and oesophagus have been recorded. In the rare cases of congenital partial defect of the oesophagus a communication of its upper end with the trachea is sometimes met with (*vide* Vol. III. p. 331). Even less rarely hernia-like pouches of the tracheal mucous membrane occur, implicating one or both sides of the tube; it has been suggested that this condition is the developmental analogue of the laryngeal sac of the gorilla. Further hernia-like

bulging of the mucosa may be due to congenital defect of the cartilaginous rings, or, when lateral and occupying the lower end, to supernumerary and rudimentary bronchi (Chiari). An external visible tumour often does not appear till later in life when the bulging has been increased by coughing. It is then called an aerocele. One of us (F. S.) has described the case of a girl in whom the trachea just below the larynx was bent from right to left and again in the opposite direction lower down, so that at the site where one would expect to see the bifurcation there seemed to be some outside influence pressing the tube inwards; at this point marked pulsation was noted. The condition was associated with a defect in the cartilaginous union of the two halves of the thyroid cartilage, and with what probably was a diverticulum of the oesophagus.

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2. **Injuries.**—Incised wounds and gunshot injuries may implicate the trachea, the former being for the most part transverse, and produced in attempted suicide. Tracheotomy wounds not infrequently cause difficulty, stenosis resulting either from granulation-tissue along the tracheal incision, especially in the upper angle and when a high operation has been done, or from kinking of the tracheal wall. Various injuries of the chest, such as kicks, may result in transverse fracture of the trachea, whilst scalds from attempts to drink boiling water, or to swallow acids or other caustics which may find their way into the trachea, are possible causes of tracheal stenosis.

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3. **Inflammatory Diseases.**—The trachea is usually more or less affected in acute inflammations of the larynx or bronchi, such as laryngitis and bronchitis from whatever cause. Scalding or the inhalation of irritating vapours may produce inflammation of the tube, provided the irritant cause either finds direct access into the trachea, or that the inflammation caused higher up is propagated by extension. Infective inflammations of the tube are most frequently due to diphtheria and influenza, but they also occur in connexion with scarlatina and other infectious fevers, scleroma, and acute septic laryngitis. Phlegmonous inflammations may extend from oesophageal disease or from cellulitis around the trachea. Perichondritis of the trachea may be either the

result of an injury, or arise in the course of enteric fever, syphilis, tuberculosis, etc., when the cartilaginous rings may be bared and sometimes expectorated piecemeal.

Oedema of the trachea, apart from that occurring in perichondritis, is exceedingly rare.

The phenomena of acute inflammatory affections are the same as those seen in other mucous membranes, namely, more or less intense redness and congestion of the mucous membrane, with formation of mucoid, muco-purulent, and, in rare cases, blood-stained secretion. On a single occasion one of us (F. S.) has actually watched the escape of blood from a spot in the anterior wall of the trachea. Events of this kind, however, are certainly extremely rare, and the statement so often heard from patients, that they had bled "from the windpipe" should always be accepted with the greatest reserve, as in the great majority of these cases the real source of bleeding was probably within the chest. On the other hand it is, of course, possible that, if a tracheal ulcer erode a neighbouring large vessel, or if an aneurysm perforate into the trachea, the resulting haemorrhage may be so copious as to be immediately fatal, or the final fatal gush of blood may be preceded by one or more less copious haemorrhages. Influenza, in the experience of one of us (W. W.), sometimes seems to cause intense hyperaemia of the trachea, and occasionally to be attended with more or less free haemoptysis, reminding one of the tendency to epistaxis early in influenzal attacks.

In some of the chronic affections of the upper air-passages, such as rhinitis, laryngitis sicca, or scleroma, secretion may run down into the trachea, or the mucous membrane of that tube may participate in the atrophic process, and firm inspissated crusts in the trachea may give rise to serious inconvenience before they are expectorated.

In cases of violent scalding, inflammation from corrosive poisons, and, of course, above all in diphtheria, the exudation is not of a fluid, but of a fibrinous character, and false membranes are formed, which may line a larger or smaller part of the inside of the tube. In very rare cases circumscribed abscesses may occur. Should a tracheo-oesophageal fistula form in the case of any ulcerative disease of the trachea, there will be escape of fluid food into the trachea, manifested by cough, expectoration, and, failing this, by entrance of the fluid into the lower air-passages, and possibly by septic pneumonia ("Speisepneumonie").

In scleroma of the trachea a small-celled infiltration occurs, which may lead to considerable thickening of the walls of the tube, and not rarely to considerable stenosis of its lumen. Sometimes the whole lining of the trachea is changed into a dense indurated mass, in which the characters of the mucous membrane have entirely perished. (See article on "Scleroma of Upper Air-Passages," p. 142.) In cases of perichondritis there is occasionally a true hypertrophy of cartilaginous tissue, characterised by general or partial ecchondroses, which may give a mammillated appearance to the interior of the tube, rounded or pointed excrescences of a whitish or yellow tint occupying a larger or smaller part of its surface.

The air-passage may become considerably narrowed. Cases of this kind have been described and figured by L. von Schroetter in his excellent *Lectures on Diseases of the Trachea*, and in 1902 by Dr. Edward Law.

The symptoms of inflammatory affections of the trachea, to whatever special cause they may be due, are very much the same, and usually are masked by the more important concomitant signs of the laryngeal or bronchial affection accompanying the tracheal inflammation. Many years ago the late Prof. Stoerck of Vienna found that the anterior and lateral walls of the trachea were extremely insensitive, and that it was only on irritating the membranous posterior fourth of the tube that cough and pain were produced. Clinical experience has corroborated this result. In inflammations of the posterior wall of the trachea, and more particularly of the bifurcation, a sense of irritation, tickling cough, and pain are prominent symptoms. Of course, should the inflammation be so extensive as to narrow the calibre of the tube, or a similar result be brought about by oedema, perichondritis, or the formation of an abscess, phenomena of dyspnoea will be superadded to those of an ordinary inflammation, and it may be mentioned here at once as an important point in the differential diagnosis between laryngeal and tracheal stenosis, to which Gerhardt first drew attention, that, whilst in the former the larynx makes violent respiratory excursions during the laboured act of respiration, in tracheal stenosis, however serious and complete it may be, the larynx remains perfectly still.

Ulcers of the tracheal mucosa may be due to tuberculous or syphilitic disease, to enteric fever, or to pressure of an ill-fitting intubation or tracheotomy tube. Perichondritis is generally secondary to enteric fever, tuberculosis, or syphilis, and the tracheal rings may be laid bare, or necrose and be coughed up in pieces. Cicatricial stenosis is particularly prone to follow syphilitic ulceration and perichondritis from any cause. Phlegmonous inflammation may extend down from the larynx in cases of acute septic laryngitis (*vide* p. 117).

Various dermatoses are cited by Newcomb as having been noted in the trachea, such as herpes, impetigo herpetiformis, pemphigus, lichen ruber, erythema multiforme, angioneurotic oedema, and one case of erythema nodosum, which required tracheotomy for sudden dyspnoea.

The *diagnosis* of the various forms of tracheal inflammation will have to be made by means of reflected light, as detailed in the remarks on tracheoscopy, or by the introduction of Killian's tubes.

The *treatment* of the various forms of inflammation of the trachea in every way corresponds to that to be adopted in the analogous diseases of the larynx, to which the reader is referred. It is possible that some forms of chronic tracheal catarrh may yield to opsonic treatment, though hitherto experience is too limited for any definite expression of opinion. It may be carried out either by means of a mixed vaccin prepared from the various organisms present in the tracheal mucus, or if, as is usually the case, the dominant organism present is the *Micrococcus catarrhalis*, by a vaccin from that organism alone. One case of cure and two of improvement by means of a combined vaccin are recorded by Dr. Allen. Should acute stenosis supervene in any of these inflammations, tracheotomy may have to be performed or intubation be tried, if the stenosis is limited to the uppermost part of the tube. With regard to chronic stenoses resulting from any of the processes just described, their treatment will be dealt with more in detail farther on (see p. 339).

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4. **Syphilis of the Trachea.**—We know of no recorded case of chancre, but erythema, mucous patches, gummas, perichondritis, and cicatrices have been observed. Condylomas, which are extremely rare, appear as red, sometimes white, superficial thickenings. In one case a projecting granular mass was visible just below the vocal cords on the anterior wall. Gummatous infiltrations and secondary ulcerations are, according to Schroetter, the most frequent manifestations of syphilis in the trachea, and usually there is diffused infiltration of considerable areas rather than circumscribed localised tumefaction. It should be remembered that gummatous infiltration of the trachea may be the only manifestation of syphilis in the patient's body. Two such cases have been seen and described by one of us (F. S.). In the first case the patient complained of loss of voice and sometimes slight difficulty in swallowing, but, curiously enough, did not attach any importance to the difficulty in breathing, although this was clearly manifested by distinct stridor, even during quiet respiration. The left vocal cord was oedematous and fixed and bent outwards posteriorly. A long way down the trachea reddish projections extended into the lumen from the walls on both sides, and the calibre of the tube was thereby narrowed to such a narrow slit that it was surprising the dyspnoea was not even greater. The larynx made no respiratory excursions. Under mercury and iodide of potassium both the subjective and objective symptoms rapidly disappeared, and in six days practically no trace of the serious conditions could be detected in either the larynx or trachea. In the other case, described in 1882, a deep extensive ulcer was seen, commencing from the fourth or fifth tracheal ring downwards, occupying the entire anterior and lateral wall of the trachea, covered with thick purulent secretion, and limited above by a sharp, steep, and much-inflamed border, whilst the lower margin could not be seen. There was great inspiratory dyspnoea, loud tracheal stridor, and the larynx made no respiratory excursions.

Energetic antisyphilitic treatment effected a very speedy cure. About this time three other cases of tracheal syphilis were seen in quick succession by him, and described in the St. Thomas's Hospital Reports, but in these there were either concomitant symptoms of syphilis in other parts of the body, or signs of old syphilitic disease in the pharynx and on the skin. Conner collected and analysed 128 recorded cases, and of these he found 51 ulcers, 20 gummas, 47 scars, 13 diffuse thickenings, 8 fibrous peritracheitides. Parker reports the case of a boy, aged fifteen, in whom there was dense fibrous tissue in connexion with the submucosa, causing marked constriction of the lumen for one and a half inches above the bifurcation. Tracheotomy was performed, but the patient succumbed. The most common seat of such infiltration is the lower end of the tube, immediately above the bifurcation; the next most frequent site is the upper portion, by direct extension from the larynx; then the middle; it is very uncommon for the entire trachea to be implicated. Gleitsmann records an instance of syphilitic tracheal bands resembling "a second glottis far below the normal one. Two dark red symmetrical membranes ran horizontally from back to front of the trachea, with a very small opening between them which was wider posteriorly than anteriorly, and were localised in the region of the fifth or sixth tracheal cartilage." These bands disappeared under prolonged systemic treatment.

There is always the danger that deep syphilitic ulcers may perforate neighbouring structures, for example the oesophagus, or a blood-vessel, such as the aorta or pulmonary artery, or the vena cava, or they may take the more favourable course of opening externally. Out of 128 cases collected by Conner, 4 died from perforative haemorrhage. The danger of dyspnoea is present in the stage of infiltration, but it is the tendency shewn by healing syphilitic ulcers to form dense firm contracting cicatrices that constitutes the later and most dangerous obstruction to respiration. On the other hand, it is remarkable how very quickly and completely gummatous infiltrations and even ulcers heal under energetic antisyphilitic treatment, without leaving any traces of their existence.

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5. **Tuberculosis.**—In the late stages of pulmonary tuberculosis the trachea is often the seat of tuberculous ulceration, but this ulceration is very rarely of any importance, as the pulmonary disease in such cases is

almost invariably too extensive to admit of arrest, whilst the tracheal ulcers generally do not produce any symptoms, except, possibly, by aggravating the patient's cough. The ulcers are more commonly seen on the posterior wall; this is no doubt due to prolonged contact with sputum containing bacilli; they do not differ in appearance from the tuberculous ulcers in the larynx, and they vary in size from being small, discrete, and superficial up to the large confluent ulcers, as in a case recorded by Schrötter, in which the ulcer extended from the sixth ring right down to the bifurcation. Only very rarely does the infiltration and overgrowth of a tuberculous granuloma lead to tracheal stenosis. J. N. Mackenzie has recorded three tuberculous tumours in the trachea which assumed the form of papilloma. He also described a distinct tumour-formation covered by unbroken epithelium, and "consisting of a mass of miliary tubercles set in a vascular network of connective tissue, and exhibiting all grades of tubercular degeneration up to cavity formation." The treatment of tuberculous ulceration of the trachea is much the same as that recommended in tuberculosis of the larynx (see p. 200). But by directly applying solutions to the tracheal wall by intratracheal injections, the tenacious secretions may be aided to escape and the pain or discomfort due to ulcerations relieved. More particularly oily solutions such as eucalyptol, terebene, menthol with a little cocaine in adepsine oil, are of value. These may be injected either by passing the nozzle of a laryngeal syringe through the glottis, or by Mendel's "median" or "lateral" method of injecting the fluid against the posterior pharyngeal wall, or laterally against the lateral pharyngeal wall, while the patient continues to respire with the tongue held out. As long as the respiration is maintained the glottis remains open, and the oesophagus being closed except during deglutition the fluid is guided into the trachea through the open glottis.

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6. **New growths** of the trachea are not common; thus, Bruns could only collect records of 147 cases, and, after a careful examination of the literature, Theisen found 135 authentic cases only (excluding granulomas). Of these 89 were benign and 46 malignant. Quite recently E. Krieg, a former assistant of Bruns, has collected another 53 cases, described between 1898 and 1907.

Of *benign growths* of the trachea the following varieties have been observed:—papilloma, fibroma, adenoma, lymphoma, lipoma, ecchondroma, osteoma, and intratracheal tumours composed of thyroid gland tissue. Of these varieties, as in the case of laryngeal benign growths, fibroma and papilloma constitute almost the whole number. Papilloma is the most frequent form, usually occurring in association with multiple laryngeal papillomas, and, like the laryngeal form, for the most part in young children. They generally grow from the anterior wall, and are confined to the upper part of the trachea, varying in size from a pea to a pigeon's egg, but one of the largest, reported by Siegert, was situated close to the bifurcation. In another case the growths extended almost throughout the whole of the trachea from the larynx to the bifurcation. Fibroma occurs as an isolated polypoid growth, from the size of a pea to a hazelnut, and is usually attached to the lower third of the trachea. Adenoma usually arises on the posterior wall, originating in the mucous glands. Intratracheal thyroid tumours of an innocent nature are occasionally met

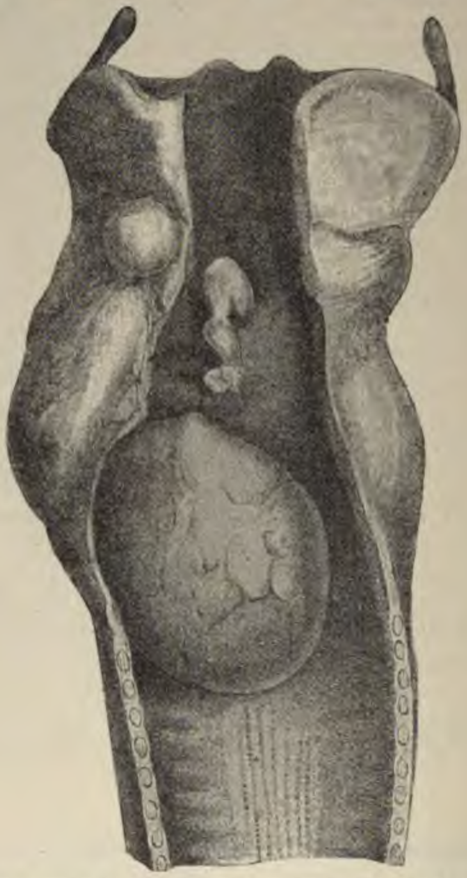

FIG. 42.—Extension of carcinoma of the thyroid gland into the trachea in the form of two pedunculated tumours which are separated from each other by a distinct interval of normal mucous membrane. Close to the base of the upper tumour is a soft tag of tissue, which probably formed the pedicle of a third tumour which sloughed away and completely disappeared during life. (From *Med.-Chir. Trans.*, London, 1893, lxxvi. Plate VI., Shattock.)

with; they appear as rounded tumours more or less completely encircling the lumen of the tube. They have been found extending up to the infraglottic space, and attached by a broad base to the lateral and part of the posterior walls of one side of the lower part of the larynx and upper part of the trachea. These growths appear to be due to a direct extension of the thyroid gland in the process of development, so that normal thyroid tissue penetrates between the cartilaginous rings as far as the mucous membrane (Paltauf, Theisen). They generally occur in association with hypertrophy of the thyroid gland proper, and all the recorded cases were between the ages of fifteen and forty.

Malignant growths occur nearly twice as frequently as the true benign neoplasms of the trachea, and primary sarcoma nearly twice as often as primary carcinoma, which is extremely rare. Of 18 cases of primary sarcoma of the trachea collected by Theisen, the larger number were attached to the tracheal wall by a broad base and had a smooth surface; occasionally they were pedunculated. A remarkable case of malignant disease of the thyroid gland, recorded by one of us (F. S.), repeatedly became pedunculated where it entered into the trachea, and on the first occasion was apparently spontaneously expectorated. Mr. Shattock suggests that the tendency of malignant growths to become pedunculated on perforating the trachea may be explained by their meeting with no resistance as soon as they extend into an open tube. The sarcomas are usually situated on the posterior and lateral wall, and develop slowly till they may come to occupy almost the whole lumen of the trachea, but do not shew any tendency to ulcerate. Whilst primary carcinoma is excessively rare, secondary carcinoma of the trachea is so much commoner that it is much more frequently seen than sarcoma. Sarcoma usually occurs high up in the trachea; carcinoma is usually situated either high up or at the lower end near the bifurcation, the middle third being a very rare situation for malignant growths. The anterior wall is very much less often attacked than is the posterior wall with its abundant supply of glands, medullary carcinoma constituting the great majority. As in the larynx, primary malignant growths of the trachea are apt to remain localised for a considerable period, and metastases in the lymphatic glands and other organs are very rare, because the patient usually succumbs to the resulting stenosis or secondary pulmonary complications before these late manifestations arise.

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7. **Stenosis of the trachea** may be due to either internal obstruction or compression from without.

A. *From Internal Obstruction, including Foreign Bodies.*—(i.) Foreign bodies; (ii.) Oedematous inflammation; (iii.) Tertiary syphilis (gumma); (iv.) Leprosy and Scleroma; (v.) Injuries and wounds; (vi.) New growths; (vii.) Penetration into the trachea of caseous lymphatic glands, abscesses, aneurysms, ingrowing thyroid glands, and other rarer forms of invasion of the tube by tumours in its neighbourhood.

B. *Compression from Without.*—The trachea may be compressed either in the neck or within the thorax. Hence we divide the most frequent causes of compression as follows:—

(α) Compression in the neck.—(i.) Enlargement of the thyroid gland; (ii.) Enlarged glands and new growths of the neck.

(β) Within the thorax.—(i.) Substernal goitre; (ii.) Enlarged glands; (iii.) Hypertrophy of the thymus gland; (iv.) Aneurysms of the aorta, the innominate, the carotids, etc.; (v.) Malignant growths in the mediastina; (vi.) Oesophageal growths; (vii.) Foreign bodies impacted in the gullet; (viii.) Abscesses in the neighbourhood of the trachea; (ix.) Cellulitis round or by the side of the tube.

A. *From Internal Obstruction, including Foreign Bodies*—(i.) Foreign Bodies.—The foreign bodies met with in the trachea may, it need hardly be said, be of most diverse kinds, such as particles of food, bones, fishbones, coins, or detached fragments of instruments, tracheal cannulas and brushes. In a sense also parts of the patient's own body, which penetrate through a perforation into the tube, such as caseous lymphatic glands or carious vertebrae, may be regarded as foreign bodies. As a rule they remain in the trachea only when their size renders it impossible for them to pass into the bronchi; but in a number of cases foreign bodies, which had entered the bronchial tubes, have occasionally, during an attack of coughing, been thrown up into the trachea again.

The symptoms caused by the entrance of foreign bodies depend, of course, upon the size and the nature of the foreign bodies. If small and round, they may cause very little in the way of symptoms, except a feeling of irritation and violent coughing when they come in contact with the posterior wall of the tube. If pointed or sharp, they may produce pain, irritation, violent cough, ulceration, and even the formation of an abscess in the neighbourhood of the tube. If large enough to cause stenosis, the degree of the latter will depend upon the size of the foreign body, and upon the position in which it is impacted. Thus, coins which change their position may lead to alternations of violent dyspnoea with comparative freedom of respiration. For the symptoms and consequences of penetration of foreign bodies into the bronchial tubes, the reader is referred to the article on Bronchiectasis (Vol. V.).

The diagnosis is to be made from the history of the case, from inspection of the tube, and from a consideration of the circumstances of the individual case. With regard to the history, however, it must not be forgotten that, particularly in children, a history of the entry of a foreign body into the air-passages is often not attainable, and exceptionally the same applies to adults, as shewn by a remarkable case reported by one of us (F. S.) years ago. A man was brought in an apparently dying state from suffocation to St. Thomas's Hospital, and immediate tracheotomy was performed to save his life. By subsequent laryngoscopic examination a large foreign body was discovered low down in the trachea. The tracheal wound was dilated, and the foreign body (an instrument used by the patient, a ventriloquist, to imitate animals' voices) was removed. The patient made a complete recovery. When the diagnosis had been made, the patient was closely questioned as to the possibility of the impaction of a foreign body, but strenuously denied this, and only after it had been successfully removed remembered that he had nearly two years previously swallowed one of his instruments. This case is a good illustration of the necessity of always bearing in mind that sudden difficulty of breathing or violent pain in the tracheal region may be due to a foreign body.

For the diagnosis by inspection the reader should refer to the introduction to this article and to the article on bronchoscopy (p. 307). With regard to the circumstances attending individual cases, sudden onset of the respiratory embarrassment should always make the practitioner think in the first place of foreign bodies, although, as in the case just described, urgent symptoms of stenosis may not appear until long after the foreign body entered the air-passages. Further, for example, when a child with the manifestations of the status lymphaticus and enlarged glands in the neck and in the throat, or with evidence of tuberculous bronchial glands, is suddenly attacked by a violent dyspnoea, the possibility that a caseous bronchial gland has perforated into the trachea, and is acting there as a foreign body, will, of course, at once occur to an experienced observer. Such contingencies might be easily multiplied. The general principle, however, is that a foreign body should always be thought of in the first place as the probable cause of the dyspnoea, when suddenly, and without other appreciable cause, symptoms of tracheal stenosis present themselves. As already mentioned, the rule that in tracheal stenosis the larynx stands still, whilst when the obstruction is in the larynx itself there are violent respiratory excursions of the larynx, is extremely useful in the differential diagnosis as to the location of the obstruction.

Treatment.—The essential point is that no foreign body, when once its presence has been definitely determined, should be allowed to remain in the air-passages, even if at the time it does not produce any serious symptoms. This principle is the outcome of the experience, that only too frequently foreign bodies, which shortly after their entry caused slight symptoms only, or none at all, subsequently produced most serious trouble. The manner in which the removal should be effected must vary according to the circumstances of the case. In the rarest instances only will mere inversion of the body and a good shaking suffice to dislodge a foreign body from the trachea, or from even lower down, and allow it to be expelled during a fit of coughing, although in a most remarkable case observed by one of us (F. S.), and described by Mr. Pitts, this fortunate result followed. Killian's direct tracheoscopy and bronchoscopy has opened up, as the steadily increasing reports of successful removal of foreign bodies from the trachea and bronchi shew, a new and most promising era for this class of accidents. For particulars of these internal operations, the reader should refer to the article on bronchoscopy (p. 299). In cases in which, for some reason or other, the direct method is not available, tracheotomy should be performed if the dyspnoea is at all urgent, and the foreign body at once removed if possible. Should there be difficulties in the way of immediate removal, it will be better to keep the trachea open by stitches in these cases, and not by introduction of a tube, in order to facilitate subsequent spontaneous expulsion of the foreign body during coughing.

(ii.) Oedematous Inflammation.—In the rarest cases only will an oedematous inflammation, unless it supervenes upon an existing stenosis, due, for instance, to malignant new growth or to a gumma in the trachea, cause genuine obstruction of the tube.

(iii.) Tertiary Syphilis.—Dangerous stenosis of the trachea is sometimes produced by the gummatous formations in the trachea, whether they appear as a solitary large gumma, or as a circular gummatous infiltration. The degree of dyspnoea depends upon two factors, (*a*) the extent to which the lumen of the tube is narrowed, and (*b*) the rapidity of the process. The more rapid the production of the stenosis, the more violent is the disturbance, and it is very remarkable what serious degrees of stenosis are sometimes borne with apparently but little discomfort by the patient, when the stenosis has come on slowly. If unrelieved, the narrowing may of course cause death, and, as already mentioned, this may be accelerated if acute inflammatory oedema should supervene upon the existing gumma. The laryngoscopic signs of tertiary syphilis of the trachea have already been described, and it only remains to emphasise here once more the necessity that the antisyphilitic treatment to be instituted should be of a very energetic character. If such be adopted, the results usually are most gratifying, and this within a very short time.

Another danger of stenosis threatened in syphilis of the trachea arises from fibrous thickening or from the formation of dense cicatrices after the healing of syphilitic ulcers. These forms may have to be dealt with by internal operation through Killian's tubes, or by tracheotomy followed by methodical dilatation of the parts, or by excision of the parts of the cicatricial tissue, followed again by methodical dilatation.

(iv.) Leprosy and Scleroma.—In both these processes, as in syphilis, thickening of the walls of the tube, general infiltration, formation of scars and of dense cicatricial tissue may occur in the trachea, which must be dealt with in the same manner as has just been recommended in syphilitic stenosis. The diagnosis of the nature of the stenosis in such cases is easy, because scleroma and leprosy hardly ever appear in the trachea alone, being almost always associated with characteristic changes in the larynx and elsewhere, from which the nature of the tracheal disease can be safely diagnosed.

(v.) Injuries and wounds to the trachea, other than those inflicted for surgical purposes, are most frequently due to suicidal or homicidal attempts, and are for that reason frequently associated with gross lesions of the neighbouring important structures of the neck. Suicidal wounds are almost always made in the transverse diameter of the tube. In a few cases fractures and lacerations of the tube have been observed as a consequence of violent injury. In such cases, unless the lesion is immediately fatal, dyspnoea, cough, with or without expectoration of blood, and particularly subcutaneous emphysema are amongst the most prominent symptoms. If recovery takes place after any form of injury inflicted upon the trachea, further dangers are to be apprehended from cicatricial contraction of the tube at the seat of the lesion, and from granulation tumours developing in or near the scar. The latter contingency is most frequently observed after the performance of tracheotomy, when an ill-fitting tube has been worn for some length of time. It particularly occurs in the case of children, and the granulation tumour is always situated at the inner and upper angle of the wound. The treatment of these cases must correspond to the exact nature of the injury. Cicatricial contraction may be dealt with either by methodical dilatation alone, or by partial or total excision of the scar tissue, again followed by long-continued dilatation; granulation tumours must be removed, and their bases energetically cauterised, after which the tube should either be left out altogether, if the nature of the case permit this, or one that fits better be substituted.

(vi.) New Growths.—The various forms of new growths met with in the trachea have already been discussed (see p. 335). Benign growths will produce symptoms only when irritating the posterior wall of the tube, tickling cough being then a prominent symptom, or when they are large enough to produce actual stenosis, in which event more or less marked dyspnoea will be present.

Malignant growths may in addition manifest their presence by fetor of the breath, pain, purulent or sanguinolent expectoration, and by symptoms traceable to perforation of the tube and invasion of important neighbouring structures. A great advance in the treatment of benign growths has resulted from the introduction of Killian's direct tracheoscopy, as it is now possible not only to diagnose, but also to remove such growths from within. In cases in which this method should not be available, tracheotomy followed by removal of the growth from without is indicated.

In cases of well-defined malignant growths, the extent of which is limited, resection of the trachea has been quite recently advocated. Should no radical operation be possible, tracheotomy will have to be performed as a palliative measure when the dyspnoea becomes urgent. In such cases, the operation should be performed as far away as possible from the seat of the growth, so that the latter may not at an early period invade the wound.

(vii.) Penetration into the Trachea of Caseous Lymphatic Glands, Abscesses, Aneurysms, Ingrowing Thyroid Glands, and other rarer forms of invasion of the tube by tumours in its neighbourhood.—Of these possible causes of stenosis of the trachea, all of which are rare, penetration of a caseous lymphatic gland is relatively the most frequent, whilst ingrowing of the thyroid gland into the interior of the tube comes next. In the rarest cases only aneurysms or mediastinal new growths actually grow into the trachea and obstruct its lumen. The diagnosis of such cases has been much facilitated by Killian's direct method, in addition to which the concomitant symptoms of the case will have to be taken into consideration. No general rules can be laid down for treatment, and each case will have to be treated according to its own merits. Thus, it is quite conceivable that, in a case in which a caseous lymphatic gland has ruptured into the interior of the trachea, and partially or almost entirely obstructed its calibre, removal from within, either after previous tracheotomy or without this operation, may now be feasible through Killian's tube. In the case of an aneurysm perforating the trachea and obstructing the passage of air, it may be possible to introduce a soft india-rubber tube past the tumour and thus relieve the breathing. Should an abscess project into the trachea, it may be possible to open it, the patient being kept in the Trendelenburg position, so as to avoid the entrance of pus into the lower air-passages, etc. The possible contingencies are too numerous and their management in every individual case too special to allow of more than these general hints.

B. *Compression from Without.*—As already stated, the trachea may be compressed either in the neck or within the thorax.

(*a*) Compression in the Neck.—(i.) Enlargement of the Thyroid Gland.—Of all possible causes of compression of the trachea, enlargement of the thyroid gland, be it of a benign or a malignant nature, is by far the most frequent. Signs of compression may be absent in cases with very large goitres, and, on the other hand, may be extremely threatening in cases with small ones: the presence or absence of compression depends entirely upon the degree of hardness and upon the situation of the enlarged gland. Thus, a very large, conspicuous, soft, parenchymatous goitre, situated high up in the neck, need not and very often does not cause any distress in breathing, whilst even small hard adenomatous nodes situated below the sternum, or between the clavicle and the trachea, may cause compression of the trachea and most distressing respiratory embarrassment. The compression in some cases is unilateral, in others bilateral.

Again, when the enlarged gland contains several hard nodules, the trachea may be distinctly deviated in different directions at different levels, and assume an S-shaped form, or be compressed in the form of a sword sheath. In some of these cases the tracheal cartilages undergo definite softening (tracheomalacia). Often a patient with the highest degree of stenosis instinctively keeps his head fixed in one and the same position, in order to facilitate the entrance of air through the stenosed passage. Should he by any chance have to turn his head from this fixed position, kinking of the tube, owing to softening of the tracheal walls, and complete obstruction may occur suddenly. In these cases, of which several have been reported, sudden death may occur during sleep. In a number of cases of compression of the trachea by goitre, respiratory symptoms are associated with deglutitory, if pressure is simultaneously exerted upon the oesophagus. In other cases, in which the enlarged gland presses upon sensory nerves, pain in various regions of the head and trunk may accompany the tracheal symptoms; and cyanosis or collateral oedema may occur when important blood-vessels are compressed at the same time. Usually, however, the symptoms are limited to those of respiratory obstruction, and in these cases the normal voice strongly contrasts with the respiratory difficulties. Again, in a number of cases—particularly when the goitre is of a malignant nature—the recurrent laryngeal nerves are pressed upon, and abductor paralysis on one or both sides (very rarely complete recurrent paralysis) may ensue. This point is of practical importance, as already insisted upon in the section on laryngeal paralysis (see p. 276), because the practitioner, who performs a tracheotomy on account of bilateral abductor paralysis, obviously necessitating the performance of this operation, may be unpleasantly surprised if, after the operation, no relief of the breathing takes place, and he finds that there is a second stenosis, due to direct compression of the trachea, lower down. A case of this kind, reported by one of us (F. S.), many years ago, became a source of serious trouble to the surgeon who performed the operation. In some of these cases the weirdest conditions are met with; thus, in a case, the specimen from which was demonstrated by one of us (F. S.) before the Laryngological Section of the International Medical Congress of London in 1881, a patient, who had a large goitre, suffered at the same time from carcinoma of the oesophagus, which caused bilateral abductor paralysis of the vocal cords. This patient died from retrotracheal haemorrhage, during the performance of tracheotomy, and the necropsy shewed that an abscess cavity had formed between the oesophagus, trachea, and right lobe of the thyroid body; this cavity communicated posteriorly through a small opening with the oesophagus, and anteriorly through a large one with the trachea. The tracheal perforation being situated just opposite the incision into the trachea, the pilot of the cannula had penetrated straight through the trachea into the abscess cavity, and, by injuring some superficially situated thin-walled veins, had caused the fatal haemorrhage.

The appearances of tracheal compression vary according to its locality, and whether the trachea is compressed from one or both sides. In the former case a crescentic bulging can usually be seen laryngoscopically; this corresponds with the level of the compressing tumour, provided that this be not too low down for ordinary laryngoscopic examination. The projection may concern the anterior, lateral, or posterior wall, according to the circumstances of the case. In cases of bilateral compression, two projections opposite one another, or one above the other, may be visible with the laryngoscope, and in the former case the lumen of the trachea may be reduced to an extremely narrow slit. When there is also, as in cases of malignant goitre, a perforation into the trachea, the surface of the invading growth may be visible in the form of a mammillated, sometimes pedunculated (*vide* Fig. 42), and, in later stages, ulcerated tumour. Here, again, Killian's direct tracheoscopy will prove of great value. In cases in which there is at the same time bilateral abductor paralysis, the diagnosis of tracheal compression will, of course, usually be impossible until the trachea has been opened, when, by introduction of a small mirror into the tracheal wound, or by means of Killian's tubes, certainty on this point might be arrived at.

The treatment of tracheal stenosis due to compression by goitre may either consist in partial or total removal of the constricting tumour (leaving some normal thyroid tissue behind in order to prevent the occurrence of myxoedema), which, when possible, will be, of course, the preferable method; or, if the dyspnoea should be extremely threatening and the removal of the goitre not be feasible, tracheotomy may be necessary. In these cases the surgeon should always, if possible, be provided with Koenig's long flexible silver cannula, or, failing this, with a stout drainage tube, in order to get below the compressing tumour, should this be situated low down in the neck, or even be substernal.

(ii.) Enlarged Glands and New Growths in the Neck.—Any enlargement of glands, and any hard tumour of the neck, whatever its histological nature, may, when situated near the trachea, cause compression of the tube and symptoms similar to those described in the previous paragraph. The diagnosis of the nature of the tumour belongs to the domain of general surgery, and cannot be discussed in detail here. If possible, the compressing tumour should of course be removed. Should this be impossible, tracheotomy may be necessary. In the case of enlarged glands, it must be remembered that they may perforate into the trachea and cause an internal stenosis in addition to that caused by compression, and hence there will be a very urgent demand for relief.

(β) Within the Thorax.—(i.) Substernal Goitre.—In the thorax, as in the neck, pressure of an enlarged thyroid gland is the most frequent cause of compression of the trachea. The recognition of these cases is easy when the supraclavicular parts of the thyroid gland are also enlarged, but in rare instances the goitrous enlargement is confined to the lower part of the isthmus, and the diagnosis of the nature of the compression may then present considerable difficulties. Occasionally it may be possible, by directing the patient to swallow, to feel the enlarged gland, which is attached by connective tissue to the trachea and therefore rises with it during the act of swallowing, in the lower part of the neck; in other cases the goitrous nature of the tumour is only discovered in the course of an operation performed to relieve the compression. Intratracheally in this and in the following forms of constriction the picture will be similar to that described in the account of compression of the neck, except that the seat of the intratracheal bulging is much lower down, and Killian's direct tracheoscopy will be found even more useful than in cases in which the obstruction is situated higher up. The treatment is, of course, removal of the substernal goitre, when possible; failing this, tracheotomy and the introduction of a long tube should be carried out.

(ii.) Enlarged Glands, including Tuberculous Glands.—With reference to the symptomatology, diagnosis, and treatment, the reader should refer to the remarks made on intratracheal stenosis (p. 336), and compression of the tube within the neck (p. 342).

(iii.) Hypertrophy of the Thymus Gland.—The history of enlargement of the thymus gland has been a rather curious one. At one time many years ago laryngismus stridulus was very commonly ascribed to it, particularly after the paper written by Kopp in 1830, which provoked quite a literature on this topic. Later on, the notion was more or less ridiculed, and the thymus gland as a cause of respiratory difficulties practically fell into oblivion, until the recent controversies on infantile respiratory spasm again drew attention to the possibility that the enlargement of the thymus gland may be the cause of the phenomena observed in that disease. Since then a few cases have been described in which it is, to say the least, possible, that the pressure of a considerably enlarged thymus gland upon the trachea and upon the nerves of the larynx was the cause of death. The subject, however, requires still further elucidation. Should it be possible in a case of tracheal compression to determine that this is due to considerable enlargement of the thymus gland, its partial or total extirpation would of course be indicated.

(iv.) Aneurysms of the Aorta, Innominate, Carotids, etc.—Enlargement of the great vessels of the neck and of the chest may compress the trachea. In such cases the bulging of the inner walls of the tube seen tracheoscopically will shew pulsation, communicated from the compressing vessel; this will considerably facilitate the diagnosis of the nature of the compression. It should be remembered, however, that, as stated in the article on tracheoscopy (p. 329), even under normal conditions pulsation is frequently seen near the spur of the bifurcation, and the discovery of this pulsation, therefore, ought not to induce the observer to rush too hastily to the diagnosis of an aneurysm, a mistake which, to our own knowledge, has been committed more than once. In cases in which tracheal compression is really due to the existence of an aneurysm of a neighbouring large blood-vessel, the diagnosis is now greatly facilitated by skiagraphy, which will definitely shew the enlargement of the compressing vessel. The treatment in such cases should be that of aneurysm of the thoracic vessels in general: Tufnell's régime, iodide of potassium, complete rest, diminution in the quantity of fluids taken, and so forth. In addition to this, tracheotomy may become absolutely necessary, but in these cases a soft long tube should always be used, in order to avoid, if possible, erosion of the compressing vessel, for there is always the danger that this may burst into the trachea and cause sudden death by suffocation.

(v.) Malignant Growths in the Mediastina.—It is obvious that carcinoma, sarcoma, or lymphosarcoma of the anterior or posterior mediastinum may cause compression of the trachea and bronchi. In such cases, unfortunately, no improvement can, in the present stage of our knowledge, be anticipated from any form of treatment; in lymphosarcoma arsenic in increasing doses might be tried. For the purposes of diagnosis Killian's tubes and skiagraphy will prove useful. By means of Killian's tubes it may even be possible to see the invasion of the trachea or bronchial tubes by the growth.

(vi.) Oesophageal Growths.—Compression of the trachea by neoplasms of the gullet is more likely to occur when the tumour is on the anterior wall of the oesophagus, as the soft posterior third of the trachea offers little resistance to the pressure of growths in that situation. In such cases a bulging will be visible on the posterior wall of the trachea, and sometimes it may even be possible to see the growth in the act of perforation. Treatment of the tracheal compression must consist in tracheotomy, which, however, in these cases will, of course, be purely palliative.

(vii.) Foreign bodies impacted in the Gullet.—In the previous paragraph it was explained why compression of the trachea is specially likely to be exercised from behind in cases of any form of distension of the oesophagus. This applies, of course, particularly to cases in which a large bolus of meat, a tooth-plate, or a large foreign body is impacted in the oesophagus opposite the trachea. In such cases the foreign body should be removed without delay, either by internal operation or by oesophagotomy, failing which, it may be necessary to push it down into the stomach. This, however, should only be done after the failure of attempts at extraction, as the procedure, particularly in cases of very large or angular or pointed foreign bodies, is not free from danger.

(viii.) Abscesses in the neighbourhood of the Trachea.—An abscess in the neighbourhood of the trachea, be it due to the caseation of a tuberculous gland, or to a septic process, or to impaction of a foreign body, or to any other cause, may, if sufficiently large, produce compression of the trachea. If its nature can be recognised, and if it be accessible to surgical interference, it should be opened the more speedily, as it must be remembered that it may rupture into the trachea itself, and cause either sudden death by suffocation, or subsequent severe pulmonary complications. Efficient drainage should be provided after the abscess has been opened.

(ix.) Cellulitis Round or by the Side of the Tube.—In cases of so-called Ludwig's angina, or of erysipelas of the neck, or of any other disease inducing immigration of pathogenetic micro-organisms into the tissues of the neck, an excessively hard brawny infiltration of these tissues may follow, leading to compression of the trachea. This infiltration should be combated, apart from constitutional treatment—such as injection of antistreptococcic serum, if it can be ascertained that the invasion of the tissues is due to the action of that micro-organism—by the free incision of the infiltrated area in order to relieve the pressure, and, if necessary, by tracheotomy (*vide* also p. 122).

Without professing that the above list exhausts all the possibilities which may lead to stenosis of the trachea, it will serve, we trust, as a guide for the practitioner with regard to the differential diagnosis and the treatment of such forms as may be met with in ordinary practice.

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8. **Neuroses.**—Exceedingly little is known about the nervous affections of the trachea. A few observers have described cases of tracheal spasm, namely, a spasm of the unstriped muscles in the posterior segment of the trachea, which may cause some dyspnoea. Lublinski actually saw tracheoscopically a prominent longitudinal folding of the mucous membrane of that part. Landgraf succeeded in curing the dyspnoea in his case by a few introductions of the tracheal bougie. Apart from this, one of us (F. S.) has described an observation, which shews that irritation of the trachea may reflexly produce violent dyspnoea. Insufflation of the balloon of a Trendelenburg tampon-cannula, which had been introduced into the trachea, set up most violent inspiratory and slightly less violent expiratory dyspnoea. During inspiration a whistling noise like the whistling of a steam engine was heard. Deep cyanosis quickly ensued, the veins of the face and neck swelling up like cords. The moment the tap of the tampon had been opened to let out a little air, the dyspnoeal paroxysm disappeared, just as suddenly and completely as it had been produced. Very likely the occurrence of such a dyspnoea must be looked upon as a reflex

phenomenon, due to sudden irritation of the tracheal branches of the pneumogastric nerve; a few other observers appear to have had similar experiences. Other nervous affections of the trachea are unknown.

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P. WATSON WILLIAMS.

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F. S.
W. W.

IV.—DISEASES OF THE EAR

Methods of Examination and General Semeiology.

General Therapeutics.

Diseases of the External Ear.

Diseases of the Tympanic Membrane.

Acute Inflammation of the Middle Ear.

Chronic Suppuration of the Middle Ear, and Suppuration of the Labyrinth.

Intracranial Complications.

Eustachian Obstruction and Chronic Middle-Ear Catarrh.

Otosclerosis.

Affections of the Labyrinth.

Deaf-Mutism.

Aids to Hearing.

METHODS OF EXAMINATION AND GENERAL SEMEIOLOGY

By Thomas Barr, M.D.

Methods of Examination

These are conveniently described under the following headings:—I. Methods of testing the hearing power and the static sense. II. Examination through the external auditory meatus. III. Examination through the Eustachian tube.

I. Methods of Testing the Hearing Power and the Static Sense.—Defective hearing, being one of the most common symptoms of ear disease, it is necessary to employ means for testing and comparing the hearing power of a patient. The tests are applied (A) by air conduction of sound, and (B) by bone conduction of sound.

A. **By air conduction** it is usual to employ (1) simple or mechanical sounds, and (2) speech.

(1) *In testing by simple sounds*, the most convenient are the tick of a watch, a special acoumeter, tuning-forks, and Galton's whistle. In testing with the tick of a watch the distance in inches at which the particular watch-tick is normally heard forms the standard of comparison; thus, if the normal hearing distance be 40 inches, and the actual hearing distance 20 inches, the condition would be expressed by the fraction $\frac{20}{40}$; if heard only on slight contact, $\frac{C}{40}$; on pressure, $\frac{P}{40}$; and if not heard even on pressure, $\frac{0}{40}$. The room should be as noiseless as possible, and each ear must be tested by itself, the other one being carefully closed by pressing the tragus into the meatus. The eyes are at the same time shut or covered. A measuring rule should be used, but it must not be touched by the watch, which is held parallel with the auricle, at first beyond the hearing limit, and then brought gradually nearer to the ear until the patient is confident that he hears the tick. The test should be repeated once or twice, as we sometimes get contradictory statements, partly due to imagination, although there is apparently in some forms of deafness an uncertain zone of hearing, indicated by the difficulty in deciding the exact moment when the tick is heard. The most common acoumeter employed is Politzer's, consisting essentially of a percussion hammer, which is raised to a fixed

height and allowed to fall on a steel cylinder, producing a sharp and uniform click, heard about 16 yards off. This acoumeter is chiefly useful when the deafness is such that the tick of a watch cannot be heard.

The tuning-fork most suitable for testing the hearing by air conduction (quantitatively) is $C^1 = 256$ vs. It is made to vibrate by striking the knee with the flat of one of the prongs, or when employing a high-pitched fork, striking a piece of wood, covered with a thick layer of cloth, with a uniform strength of stroke. The prongs are held near the orifice of the ear, but not touching the skin, until the sound has died away, when the fork is quickly removed to the patient's good ear, or to the ear of the examiner, and the difference in time noted during which the sound continues. The tuning-fork is much more useful, however, in determining the patient's power of hearing the *pitch* of notes (qualitatively). For experimental purposes, or for the exhaustive examination of the hearing, an extensive series of tuning-forks is employed, ranging from the lowest to the highest notes, but for ordinary purposes five tuning-forks are sufficient, extending from $C = 128$ vs. (Fig. 43) to $C^4 = 2048$ vs. Lower forks such as those with vibrational numbers of 64 and 32 are in some cases clinically useful. Useful work, however, may be done with three, one for the lower tones of the scale ($C = 128$ vs.), one for the medium ($C^1 = 256$ vs.), and one for the higher ($C^3 = 1024$ vs.). In using these the time during which a high-pitched fork is heard should be compared with a low-pitched one—using an equal strength of stroke each time and the difference noted. It is pretty well established that defective appreciation of the lower tones usually points to a defect in the conducting structures, and that failure to hear the higher notes of the scale generally indicates a defect in the nerve structures. If there be defective perception of both low and high tones, but good perception for medium ones, the likelihood is that both the conducting and the nerve structures are implicated. It may be said that the results are more reliable in cases in which the deafness is very pronounced and one-sided. In considering the significance of defective hearing of high notes, it is important to remember that elderly patients usually hear the high notes badly, and the same holds good with those who work amid noisy surroundings. Bezold, by testing deaf-mutes with an extensive series of tuning-forks, found that many heard certain tones or possessed a limited range of hearing, even to the extent of several octaves (islands of hearing), while there might be gaps in the scale, extending from a half to three and a half octaves. More frequently these gaps were in the lower tones, less so in the upper. Bezold considered that the perception of the range of tones from B^1 to G^2 is essential in order to hear speech even partially.

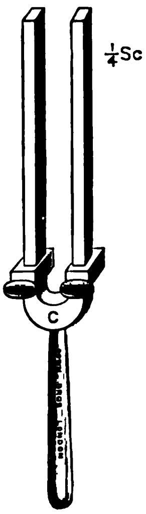

FIG. 43.—Tuning-fork with clamps.

Galton's whistle (Edelmann's) is probably more useful than the tuning-fork as a means of testing the power of hearing the high tones, and of thus determining the presence or absence of disease of the nerve structures (Fig. 44). The tube of this whistle has a very fine bore, furnished with a movable piston, by which it can be shortened or lengthened, thus raising or lowering the pitch. A small india-rubber ball is attached, by which the air is blown into the tube, and the note produced. The distance of the piston from the lower end of the tube is marked with a millimetre scale from 0 to 25 mm., and, by referring to a table which is provided, we can tell the exact number of vibrations of the particular note. In normal hearing this whistle may be heard when yielding a note as high as 50,000 vibrations in the second. If the high notes, say over 20,000, are not heard, there is probably a nerve defect.

König's rods may also be used for testing the upper limits of hearing. They consist of a series of steel cylinders, of graduated lengths, suspended by threads; by tapping with a steel hammer they yield a wide range of tone, up to 50,000 vs. They are, however, comparatively seldom employed in the examination of patients.

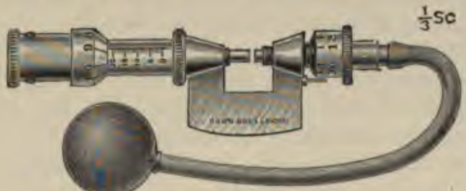

FIG. 44.—Galton-Edelmann's whistle.

It is to be noted that the results of these tests with tuning-forks and Galton's whistle must always be considered in conjunction with other points, such as the relation of air to bone conduction, the clinical symptoms, and the history of the case. It is also right to say that, in carrying out these tests, much patience is required, and their value depends a good deal on the intelligence of the patient and also upon his musical appreciation.

(2) *Testing by Means of Speech.*—This mode of testing is important, because many patients hear a watch much better proportionately than speech, and vice versa; the power of hearing the tick of a watch may thus be no criterion of the patient's power of hearing speech. Patients are being constantly tested in this way through ordinary conversation at home and in daily life, and conclusions drawn. In testing the patient's power of hearing speech, it is particularly necessary that his eyes should be covered or turned away, so that the face of the speaker may not be seen, and the opposite ear must be carefully stopped. Obviously a quiet room is very desirable.

A whisper is usually more suitable than ordinary speech, and the examiner should cultivate a low uniform whisper. The value of this test is lessened by the different degrees of intensity and pitch with which different persons speak and whisper, and also by the difficulty which the same person has in maintaining the same loudness at different times. To avoid guessing, it is well to employ single words, the patient repeating them word for word, after the examiner; and the distance should be recorded as in the case of the watch. Consonant sounds are not heard so well by deaf persons as vowel sounds, which, being richer in tone, are generally heard, except in extreme deafness; this is partly the reason why singing is heard much better than speech by the deaf. Consonant sounds are frequently mistaken—for example, "marble" may be heard as "gargle," "grocer" as "broker," or "man" as "fan." Oscar Wolf's researches shew the distance at which the vowel and consonants can be heard when loudly sounded. He found that if they are pronounced as in German, and the distance is expressed in paces, the highest is A (Ah) which is heard at a distance of 360 paces, whilst the lowest and feeblest is H aspirate, heard only at 12 paces. Between these extreme limits Wolf found the following in order from above downwards:—o, ei, e, i, eu, au, u, sh, m, n, s, f, k, t, r, b, g. We need not, however, lay too much stress on these results in regard to distances, as the intensity and distinctness of a whisper varies in different persons. They must be kept in view, however, in selecting suitable words for testing. The same words should not be repeated on different days, as familiar words are heard better than those not well known; for example, the words of a foreign tongue are heard with very much more difficulty than those of the mother tongue. So also friends and others closely associated with the deaf person are much more easily understood than strangers. In severe deafness it is necessary to employ a conversational or even a loud-spoken voice, and it is to be remembered that the hearing of a whisper is not always a true indication of the power of hearing ordinary speech. When very deaf, the patient's capacity for hearing through a conversation-tube should also be tested. Testing by numbers does not seem to be trustworthy. It is well in some cases also to test both ears together with conversational and whispered speech, as being the usual condition of hearing in daily life, the patient with his eyes closed facing the examiner. According to Oscar Wolf, the tones of speech have a compass of 8 octaves, from $C^2 = 32$ vs. to $C^5 = 4096$ vs. The highest is represented by S, the lowest by R lingual. Groups of words have been arranged by Wolf and others to represent the various notes, and it is asserted that by testing with these we are helped in the differential diagnosis of labyrinthine disease.

When testing a young child in whom complete deafness is suspected, we may employ the sound of a bell, a loud whistle, a tuning-fork, clapping the hands, or a very loud voice, taking care that the child's face is turned away from the source of sound. We must not produce a sound by stamping on the floor or knocking on the wall or door of the room. If a silent tuning-fork be applied to the forehead or near the ear, the child's features will probably remain unaltered, but if applied afterwards while vibrating, the child's smile or cry of surprise will frequently shew that he hears the note, because there is usually some degree of perception of sound in deaf-mutes.

Simulated Deafness.—When the person does not profess to be totally deaf, it is easy to detect the malingerer by testing the hearing power while he is blindfolded, when his contradictory replies will reveal the true state of matters. When he professes total deafness careful watching is necessary to expose well-planned deception. Such expedients as observing if loud speech awaken the individual out of sleep, or suggesting in his presence painful modes of treatment, or noting the effect of ordering him "to go as he is unfit for work" may be tried. In a case of a young woman under my care the simulation was discovered by observing that one afternoon she sang the identical song, an uncommon tune, which had been sung in her presence by a servant on the same day. When one-sided deafness is feigned it is a good plan to use a double-tube stethoscope, with the tube for the hearing ear plugged. When it is spoken into, the person will probably say he hears although we know that he cannot. If the tube is now removed from the hearing ear and the latter closed with the finger he will profess no longer to hear, knowing that the hearing ear is shut, while the tube of the stethoscope is only in the ear in which he pretends to be deaf.

B. **In testing the hearing by bone conduction and comparing it with air conduction,** a vibrating tuning-fork is usually employed, and the one most useful for this purpose is of the pitch $C = 128$ vs.; but $C^1 = 256$ vs. is also required. The end of the handle is preferably flat and should be applied with moderate pressure to the surface of the head. The harmonics, which must be excluded, are destroyed when the ends of the prongs are grasped by metal clamps, while at the same time the pitch is lowered by an octave. There are three methods usually employed, namely, Weber's, Rinne's, and Schwabach's.

Weber's test is most useful in one-sided deafness, and consists in placing the sounding-fork, $C = 128$ vs. preferably, in contact with the middle line of the head, such as the vertex, the forehead, the bridge of the nose, or the middle of the chin, and ascertaining in which ear the sound is heard more loudly. In a general way it may be said that, if the sound of the tuning-fork be distinctly referred by the patient to the affected or deafer side (positive effect), the cause of the deafness is in the external or middle ear, such as ceruminous obstruction, exudative middle-ear catarrh, excessive tension of the tympanum, swelling of the tympanic lining, or fixation of the stapes. On the other hand, if the sound of the tuning-fork be clearly referred by the patient to the normal or less affected side, the fault is probably in the labyrinth or auditory nerve. The patient, being biased by the thought that he *should* hear the tuning-fork better on the good side and worse on the deaf one, will often at once say that he hears it better on the normal side; but, if he be previously cautioned, we shall generally succeed in getting accurate information.

Rinne's test consists in applying the vibrating tuning-fork, with moderate and uniform pressure, to the base of the mastoid process, and, after the sound has completely died away, removing it rapidly so that the flat of the prongs shall be opposite and near the orifice of the ear, taking care that the tone is not heard by the other ear. If the sound then becomes again audible, the result is said to be positive (+ R), the condition in normal hearing. When, on the contrary, the sound remains inaudible after the transference, the result is said to be negative (– R), bone conduction being in excess, as in many cases of disease. The opposite order may be adopted, using the tuning-fork first by air conduction and transferring it to the mastoid the moment the tone has ceased to be heard. The time in seconds during which the tuning-fork is heard by bone conduction, after it has ceased by air conduction, or by air conduction after it has ceased by bone conduction, should be observed and noted, and the longer these periods the more trustworthy is the test. It may be said in a general way that, when, in a case of marked deafness, the bone conduction predominates over the air conduction (– R) the obstacle to hearing is in the external or middle ear, and that when, on the contrary, the air conduction predominates (+ R) we may infer that the labyrinth, or the auditory nerve, is the seat of the mischief. These results must be taken in connexion with other tests. It must also be noted that Rinne's test generally gives a positive result in a person over fifty-five years of age, and also in one who has worked amid noisy surroundings for a long time.

By *Schwabach's test* we compare the patient's bone conduction with our own, provided the latter be normal. If, the moment the patient has ceased to hear the sounding-fork on his mastoid process, we quickly transfer it to our own and we fail to perceive it, or hear it only slightly, the indication is in favour of a normal condition of the nerve structures. If, on the other hand, we hear it clearly and for some time after the patient no longer hears it, we may conclude that there is nerve impairment.

In deciding as to the significance of these tests, it must be remembered that there often exist in the same person affections of both the conducting and nerve structures. There may be, for example, an undoubted affection of the conducting structures, with a positive Rinne. On inquiring, however, we may find that the person had worked for a long time amid loud sounds, thereby damaging his auditory nerve structures, enfeebling his bone conduction, and so neutralising the effects on the bone conduction of the disease in the conducting structures. Or a patient may have clear signs of nerve mischief, and yet there may be a negative Rinne, which, after the removal of a plug of cerumen, may become positive. The plug so reinforced the bone conduction that the effects of impaired nerve structures were more than neutralised. We sometimes find that, while Rinne's test gives a positive result, Weber's may refer the sound to the affected ear; in such a case probably the latter is the more reliable. Not infrequently it is found that Rinne's test yields a positive response to a higher pitched tuning-fork, say one of 256 vs., and a negative one to a lower, say one of 128 vs.; this is partly explained by the rule that low notes are badly heard by air conduction in affections of the conducting structures, while higher ones may be well heard.

The bone conduction is sometimes tested with *the watch*, which should be applied first to the temple, then to the mastoid process while the ears are closed. As the sound of even a loudly ticking watch may not be heard by bone conduction, although the perceptive power is unimpaired, the value of this test is very limited. If, however, a weakly ticking watch is clearly heard, the indication is in favour of a sound nerve.

Gellé's experiment consists in applying the sounding tuning-fork to the vertex or the mastoid surface, while compressing the air in the meatus by Siegle's speculum. If the sound remain unaltered (negative result), many authorities consider that there is evidence of fixation of the stapes. If, on the other hand, the tone be diminished during the compression (positive result), or if giddiness be excited, there is probably a movable stapes. This is rather a troublesome test, and the answers of patients are not always reliable.

It must be admitted that the value of these tests, as applied to bone and air conduction, is a good deal impaired by anomalies and exceptions, so that one test may seem to contradict the other. If, however, the results of the tests of Weber, Rinne, and Schwabach (the so-called Bezold's triad of symptoms) are in agreement, a fairly safe conclusion can be drawn, and we are greatly aided in the differential diagnosis of disease of the conducting structure from disease of the nerve structures. No doubt the inapplicability of these tests to persons over fifty-five years of age very much limits their value. In forming a conclusion we are, of course, greatly helped by the use of high- and low-pitched tuning-forks and Galton's whistle, as well as by the clinical symptoms and the history and causation of the disease.

Testing with the Galvanic Current.—It need only be said that the diagnostic value of the galvanic current in ear disease is still *sub judice*.

Testing the Static Sense.—It is now well established that vertigo, or disturbance of equilibrium, is frequently connected with aural disease, and it is therefore important to test the "static sense" or the state of equilibrium. When manifest ear disease exists, and the patient complains of giddiness, we should first make sure that he understands what giddiness really is, and ascertain if deafness and tinnitus are present, also if there be vomiting. We should also inquire whether the giddiness has sprung out of an acute Menière's attack, or if it has arisen in the course of a chronic ear affection. No doubt the worst forms occur in labyrinthine affections; and therefore it is well to employ the tests already described for differentiating disease of the nerve structures from that of the conducting structures. We should also ascertain whether the vertigo is produced or increased by such processes as inflation, syringing, or the use of Siegle's speculum; or if it is excited by certain movements such as looking downwards or upwards, or turning to the right or left. We should also inquire if the sense of movement refers to the patient himself or to external objects, and if the tendency is to turn or fall *from* or *towards* the ear affected. We should also determine the absence of any ocular disturbance sufficient to account for the giddiness. By employing the following tests, both with the eyes open and shut, we may, by observing whether there is any tendency to sway or fall, determine the condition of the static sense, and, if impaired, the degree of impairment: (i.) standing on both legs with feet together; (ii.) standing on the toes; (iii.) standing on one foot, noting whether equilibrium is more disturbed when standing on the foot corresponding to the affected ear; (iv.) jumping on the two feet; (v.) walking in a straight line, observing if he reels or walks zigzag or with his legs apart; (vi.) walking with knees straight; (vii.) rotation with feet together; (viii.) rotation on one foot.

Stanislaus von Stein describes an apparatus—the goniometer—which he states is useful in testing the static and dynamic sense in aural disturbance. The patient stands upon a foot-board—an inclined plane—which can be raised or lowered at an angle, by means of a hinge arrangement, like a trap-door. There is a quadrant with a scale to measure the angles of movement and thus determine the amount of elevation which leads the patient to fall forward or backward, the test being conducted both with the eyes open and shut. It is asserted that in this way it is possible to express in figures the degree of statical disturbance and to trace the changes as time goes on.

Oscillatory movements of the eyeball (*nystagmus*) may be associated with disturbance of equilibrium due to ear disease, especially the chronic purulent forms. If nystagmus be present, the examiner should observe whether the movements of the eye are horizontal, rotatory, or vertical, corresponding with the planes of the semicircular canals; also, if they are excited or made worse when the eyes are turned to the side of the affected ear, or to the opposite side; likewise whether syringing or manipulating the interior of the ear, such as by pressure with a probe, produces or aggravates the nystagmus.

II. EXAMINATION THROUGH THE EXTERNAL AUDITORY MEATUS.—The external parts, including the auricle, the outer opening of the ear, the mastoid surface, the glandular structures, and the facial aspect do not require any special methods of examination. Simple inspection and palpation are sufficient. Examination through the external meatus, however, demands special appliances and yields important information. It is usually carried out with the aid of a speculum and reflecting mirror. The aural speculum is a funnel-shaped tube about 1½ inches in length, made either of German silver or of vulcanite. The former is more commonly used, especially that known as Gruber's, which has an oval lumen and a serrated outer edge. Four sizes are necessary in order to fit the external canals of different persons and at different ages. Light is projected into the speculum by means of the concave mirror introduced by von Tröltsch. This has usually a diameter of about two and a half inches, a focal distance of from three to five inches, and is furnished with a small aperture in the centre. Whilst the mirror may be used with a handle screwed on to the back, it is more conveniently supported in front of the eye of the operator by means of a head-band, as in the case of the laryngoscope; some surgeons use a spectacle frame, others a metallic head-spring. Any such arrangement admits of the use of the right hand for manipulation. For persons with faulty refraction a proper lens may be fitted behind the aperture in the mirror, or suitable spectacles should be worn. Good light is essential. Daylight, especially that reflected from white clouds or a white wall, affords very good illumination, and the direct rays of the sun shew very minute changes on the membrane, and may even partially illumine the interior of the tympanum. It is, however, generally necessary to employ artificial light. Either lamp-light or gas-light (both of which impart a reddish-yellow tint to the parts) will serve the purpose; and in the latter case an Argand burner or a Welsbach incandescent burner (furnished with a glass funnel) is suitable, supported by a wall bracket or table standard. An incandescent electric lamp with whitened glass, 32 candle-power, is frequently employed, and has the advantage over gas or oil of being cooler, and therefore more comfortable to the patient. An electric forehead lamp, fixed to the head, may be used, or may be attached to the front of the mirror. The oxy-hydrogen limelight is sometimes employed for delicate operative work. A portable oil or spirit lamp (Baber's) is useful for the bedside. Candle-light may suffice with the practised observer, in the absence of a stronger light.

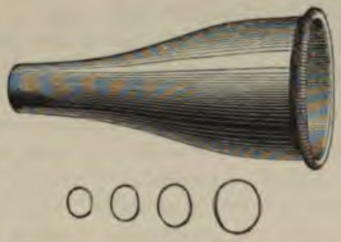
FIG. 45.—Gruber's metallic speculum.

Mode of using the Mirror and Speculum.—The examiner, having the mirror adjusted in front of the right eye, moves his head sufficiently near to the patient's ear (which should be in shadow), manipulating the mirror so as to direct the reflecting surface towards the light, which is reflected on to the meatus. The auricle is now held between the left index finger in the concha, and the middle finger behind the auricle, and gently pulled upwards and backwards in order to straighten the canal, while the right thumb presses the tragus forward. In this way, without a speculum, we may see well into the meatus, and even as far as the tympanic membrane. We may thus detect excess of cerumen, pus, or mucus, granulation tissue or a polypus, eczema or a furunculus, or stenosis. It is important to inspect the meatus in this way before introducing the speculum which might readily conceal such a condition as eczema of the orifice, or excite great pain if there should be a furunculus. The auricle being held backwards in the way described, the speculum (which should be as wide as possible and warmed) is held by its outer edge with the right hand and passed carefully in with a slight rotatory movement, until the membrane is clearly exposed by the light projected into the speculum. The speculum is moved about between the left thumb at its lower edge and the index finger in the concha, so that the examiner may observe the different parts of the meatus and the tympanic membrane. In order to see the parts properly it is often necessary to remove obstructions in the meatus, such as purulent secretion, a collection of cerumen, epidermic scales, or hairs. In removing purulent secretion or ceruminous masses, syringing is usually required with, it may be, previous softening by means of a warm solution of bicarbonate of sodium in glycerin and water. After syringing, the interior of the ear should be carefully dried with cotton wool applied on a cotton-holder—a piece of iron or wire with both ends roughened, or in the form of a screw, round which the cotton wool is firmly wound into a cylindrical shape. Epidermic scales or small particles of cerumen may be removed by suitable forceps or pushed aside with a probe, but only while light is reflected into the meatus. Inflammatory swellings of the meatal walls may temporarily obstruct the view. A more permanent difficulty is presented by an exostosis, or by an unusually prominent antero-inferior wall, which, in certain cases, may admit of the postero-superior quadrant only of the membrane being seen. The speculum suffices to push aside any hairs.

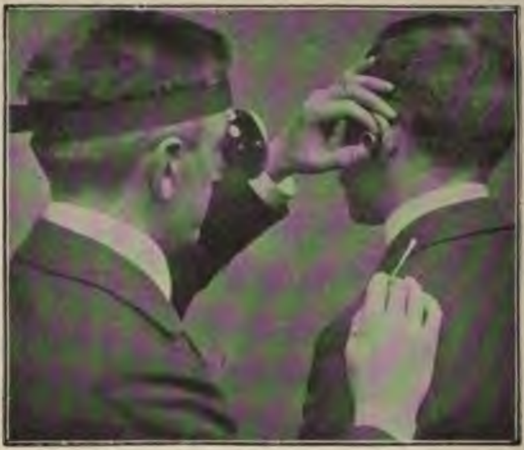

FIG. 46.—Examination through the external meatus with forehead mirror and speculum.

Siegle's pneumatic speculum consists of a vulcanite speculum screwed on to the end of a vulcanite cylinder, which is closed at the other end by an obliquely placed plate of glass. A perforated peg is fixed over an aperture in the side of the cylinder, and to this is affixed an india-rubber tube, furnished at its other end with a Delstanche's pump, an india-rubber ball, or a mouthpiece. In order that the speculum may fit air-tight into the meatus it is covered with a small piece of india-rubber. When using the mirror and speculum, we observe the behaviour of the membrane while the air in the meatus is rarefied, or alternately rarefied and condensed by the action of the pump, rubber-ball, or mouth. During rarefaction the membrane is seen to move outwards, especially above and behind. When there is stiffening of the membrane or adhesion to the inner tympanic wall, there is no movement or a very slight one. On the other hand, a cicatrix or an atrophied portion will bulge outwards. In this way also a small perforation may be located by observing aspirated secretion at a point in the tympanic membrane. The amount of secretion may sometimes be so great as to point to a source larger than the tympanic space. Furnished with a pump, propelled by an electromotor, this instrument, or a modification of it, is now much used in treatment (pneumo-massage of the tympanum, *vide* p. 385).

While examining with the speculum and mirror we note (1) the condition of the *external canal*, such as its width and curvature, the colour of its lining, the quantity and character of the cerumen, the presence or absence of mucous secretion, epidermic material, polypi or granulation tissue, a hyperostosis or exostosis, furunculi, or cutaneous thickening or swelling; (2) *the tympanic membrane or drum-head* (Fig. 2, Plate XIII.). In health this structure presents by daylight a pearl-grey shining surface, darker in front of the handle of the malleus, with a general concavity outwards, and such an obliquity that the upper and back part is nearer the eye of the observer and more easily seen than the lower and front part. Its most prominent feature is the *handle of the malleus or manubrium*, a bony ridge, varying in size in different persons, but broader at the upper end, extending from the antero-superior pole of the membrane downwards, backwards, and slightly inwards to a point somewhat below the middle, where it ends in a grey spade-like expansion—the *umbo.* From the obliquity of the membrane the posterior side only of the handle is seen, the anterior being concealed and in shadow. The upper end of the handle appears as a small rounded white knob, *the short process.* Extending from the lower end of the handle, downwards and forwards, there is usually a bright reflection termed the *cone of light*, with the apex at the umbo. This varies in different persons, and is often broken up into several parts or reduced to one little spot; these varieties have but little significance. From the short process a narrow, more or less distinct fold, passes backwards—the posterior fold—and a less distinct one in front, the anterior fold. The small area above the short process and these two folds is known as the *membrana flaccida*, or *Shrapnell's membrane*, which occupies the "Rivinian" segment, and varies in size in different persons. When the tympanic membrane is specially transparent (Fig. 3, Plate XIII.), we may see the long process of the incus through it as a whitish streak, slightly behind and parallel with the upper part of the handle, and from its lower end the posterior crus of the stapes may be observed passing backwards, forming with the incus an elbow-shaped appearance;

even the tendon of the stapedius may in some cases be discernible. Occasionally there is also visible below and behind a dark semilunar area —the depression of the fenestra rotunda on the inner tympanic wall; whilst, behind the umbo, a yellowish-grey colour may be reflected from the promontory.

III. EXAMINATION THROUGH THE EUSTACHIAN TUBE.—In a complete examination of the ear, the nasal passages and the naso-pharynx should always be included. This involves anterior and posterior rhinoscopy, which are described elsewhere (see p. 3). We shall here limit ourselves mainly to the methods of examining by means of inflation of the middle ear and its diagnostic significance; its therapeutic importance will be considered elsewhere. There are two methods employed by the surgeon, namely, *Catheterisation* and *Politzerisation*, and one effected by the patient himself—*Valsalva's experiment.*

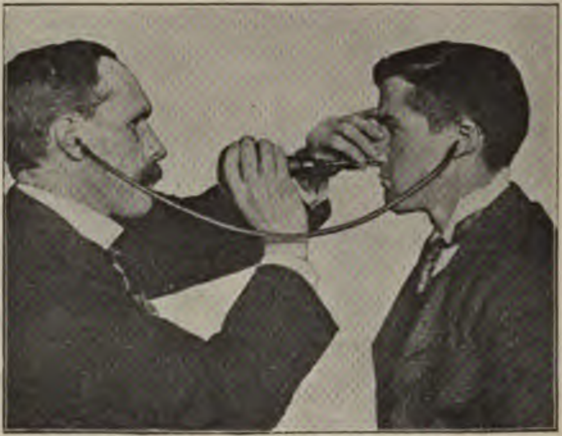

FIG. 47.—Inflation by catheterisation of the middle ear.

In inflation by **Catheterisation** a suitably formed tube — the Eustachian catheter—is passed through the inferior meatus of the nose into the pharyngeal mouth of the Eustachian tube, and then a current of air is forced through it. The catheter (Fig. 48) is made either of German silver or of vulcanite, curved at the inner end in the form of a beak, the point of which is intended for insertion into the mouth of the Eustachian tube, whilst the outer end is widened so as to fit on to the mouthpiece of an india-rubber bag. The surgeon should be provided with several catheters, differing in thickness and in length and curvature

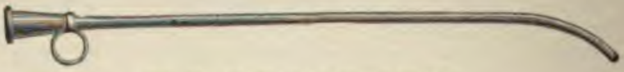

FIG. 48.—Vulcanite Eustachian catheter.

of the beak. A ring is attached near the outer end, corresponding with the concavity of the curve, so that when the catheter is introduced the position of the ring indicates the position of the beak. Vulcanite catheters are more commonly used than the metallic ones. They are not so readily injured by fluids injected through them, and they are probably less unpleasant to the patient, whilst by their elasticity obstructions are more easily evaded. The metallic ones have, however, the advantage of being capable of sterilisation by boiling. Before introducing a catheter it is well first to inspect the nasal passages in order to ascertain if there be any obstruction and the nature of it, and we should also blow air through the catheter to test its permeability. In sensitive patients a 10 per cent solution of cocaine, applied on cotton wool or as a spray, renders the operation less unpleasant and less difficult. The patient should be seated and his face well exposed to light, while the back of the head should rest against some firm support.

The first stage consists in passing the instrument through the inferior meatus till the point of the beak is felt to be in contact with the posterior wall of the naso-pharynx. While the four fingers of the surgeon's left hand rest on the patient's forehead, his thumb gently presses up the tip of the nose. The catheter is held like a pen, very lightly and with every care to avoid pain, while the point of its beak is placed within the nostril, in contact with the floor of the nasal passage, which is under the level of the inferior edge of the nasal entrance. It should now be pushed carefully but not too slowly along the inferior meatus, with the point of the beak kept in contact with the floor until it touches the hard posterior wall of the naso-pharynx, when the outer part of the catheter should form a right angle with the face. If the catheter has slipped into the middle meatus—the most common mishap of the beginner—it will form an obtuse angle with the upper part of the face and will cause pain, and the next stage—the turning movement—is rendered impossible. The most common form of obstruction, rendering the passage of the catheter difficult, is a deflection or spur on the septum, or an abnormally large or projecting inferior turbinal, or, still worse, both of these combined. In these obstructions the use of cocaine is of great service, increasing the lumen of the meatus by causing the tissue to shrink and lessening sensation. By a little manipulation, or by the use of a thinner catheter or one with a smaller curve, the surgeon is generally able to overcome any difficulties. If he fail, the catheter may be introduced through the opposite nostril; but then a catheter, having a long beak and a pretty strong curve, is required.

In the second stage the point of the catheter is moved from the posterior wall of the naso-pharynx to the mouth of the Eustachian tube. By one of the most common methods (Bonnafont's) the point of the catheter is turned outwards into a hollow in the lateral wall of the naso-pharynx, the fossa of Rosenmüller, then withdrawn till its point is felt to pass over the rounded firm projection of the postero-superior border of the mouth of the Eustachian tube. By another convenient plan (Loewenberg's) the point of the catheter is turned towards the opposite side, then withdrawn, until the beak is felt to hook round the posterior edge of the nasal septum. The catheter is then held between the thumb and index finger of the left hand, while the other three fingers rest on the bridge of the nose and the forehead; supported in this way the catheter is rotated downwards and outwards, and then the point is usually at the mouth of the tube. These two methods have well-marked anatomical guides, namely, the projection behind the mouth of the Eustachian tube, and the posterior edge of the nasal septum. A less reliable method is to withdraw the catheter half an inch from the posterior wall of the naso-pharynx, or till the beak is opposed by the soft palate, and then turn the point outwards towards the mouth of the tube. The point of the catheter is helped into the mouth of the tube by gently pressing the catheter against the nasal septum, and when the catheter is properly introduced the ring is directed towards the outer canthus of the same side. There is sometimes difficulty in turning the beak of the catheter owing to lack of space in the naso-pharynx, or more frequently from spasmodic contraction of the pharyngeal muscles. The smallness of the space may be met by a thinner and more slightly curved beak, whilst the muscular spasm generally passes off when the patient inspires deeply through the nose. Coughing, retching, or sneezing may be excited; but these usually pass off by letting go the catheter. There is occasionally slight bleeding, but this is rarely more than a stain on the beak. Even the experienced surgeon cannot always satisfy himself by touch alone that the catheter is properly introduced, and he must prove it by forcing air through the catheter into the middle ear while using the auscultation tube (third stage).

During the *third stage*, that of inflation, the catheter is kept in situ by holding it between the thumb and index finger of the left hand, while the hand is steadied by resting the other three fingers on the bridge of the nose and forehead. The catheter inflating bag is a pear-shaped india-rubber bag of eight ounces capacity furnished with a tubular mouthpiece, which should fit accurately into the outer opening of the catheter. This bag may be conveniently held in the left armpit of the surgeon while the catheter is being introduced. The mouthpiece being carefully placed in the outer orifice of the catheter, the bag is firmly compressed laterally between the four fingers and thumb so as to force the air into the catheter. Great care must be taken to avoid pushing the catheter inwards during the compression of the bag. Before relaxing the hand and allowing the bag to fill with air, the mouthpiece should be withdrawn from the catheter. Three or four compressions of the bag are generally sufficient, and after each, the bag, if it have no valve, should be removed while it fills with air. If the patient swallow during inflation, the air is usually heard to pass in more freely owing to the contraction of the tensor palati muscle. To avoid startling the patient the first inflation should be gentle, and if there is much resistance to the entrance of air we must not use force, but rather readjust the catheter. By using force the mucous membrane may be injured, and air driven into the submucous tissue, causing emphysema of the neighbouring parts, such as the soft palate, uvula, or neck. With ordinary care this will be an extremely rare accident, and can never prove a serious complication. After use the catheter should be thoroughly cleansed by syringing with very hot water, and placed in a 5 per cent solution of carbolic acid till again required, when it is washed with very hot water before use. Metallic catheters should be sterilised by boiling. When a patient requires repeated use of the catheter it is well to reserve one for his use. In syphilitic cases this is imperative.

Inflation by Politzerisation (Fig. 49).—We pass now to the important method of inflating the middle ear, named after its discoverer, Prof. Adam Politzer of Vienna. The patient is seated, and takes a little water into his mouth. The end of a tube connected with an air-bag (similar to the catheter bag, but smaller for children), which is held in the examiner's right hand, is introduced with the left hand about one-third part of an inch into the outer angle of the nostril close to the floor of the inferior meatus, the nostrils being then firmly closed over the tube with the thumb and index finger of the left hand. The patient is told to swallow, and, immediately after, the bag is compressed. The naso-pharynx is closed by the raising of the soft palate in swallowing, and the compressed air in the nasal cavity generally makes its way through the Eustachian tubes into the tympana; this is facilitated by the contraction of the abductors of the tubes during swallowing. By the sense of resistance in compressing the bag we can often tell whether the soft palate has closed accurately. If there is a difficulty in making the inflation synchronise with the act of swallowing, it is a good plan to observe the larynx and compress the bag when the larynx begins to rise in swallowing. The air, of course, tends to enter the ear in which there

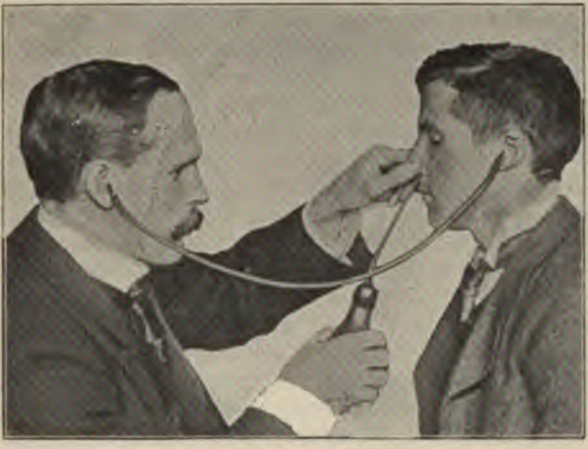

FIG. 49.—Inflation by Politzerisation.

is less obstruction in the Eustachian tube; and, if we wish to limit the effect to one side, the opposite ear is tightly closed with the patient's finger. Several kinds of nasal pieces are employed. The original form is shaped like a catheter, and is connected with the air-bag by means of soft india-rubber tubing. The part inserted into the nostril should be covered with soft india-rubber tubing; and, if it is also flattened, the tube is less likely to hurt or cause bleeding by abraiding the mucous membrane. A soft nasal piece made of thick india-rubber or in the form of an air-cushion, somewhat flattened at the side, is specially suited for children and sensitive persons. If a large olive-shaped nasal piece is employed filling up one nostril, so that only the other is closed with the fingers, there is apt to be reflux of air rendering the inflation less efficient. Mayer and Meltzer have introduced a convenient bottle-shaped bag with soft nozzle. Some surgeons use with success an ordinary india-rubber enema bag, of six or eight ounces capacity, having a piece of soft india-rubber tubing covering and projecting from its hard mouthpiece. Self-inflation can be practised with a simple india-rubber tube, the end of which is inserted into the nostril, while, with the nostrils closed, the patient blows into the mouthpiece at the other end. In all cases great care should be taken that the nose-piece is very thoroughly cleansed after use. A good plan is to have a fresh piece of india-rubber tubing with each patient. Sometimes the swallowing of water is impracticable, as in a young child, or the ordinary method of swallowing may not yield a successful result. In such cases the vigorous sounding of "Ah," or the act of crying in the case of a child, is likely to close the naso-pharynx and allow of effective inflation. The loud pronouncing of "hook," with prolonged emphasis on the "k," as suggested by Gruber, is probably still better. Blowing out the cheeks during inflation, as recommended by Holt, often proves a good substitute for the swallowing of water. Politzer suggests as an alternative method that the patient should take a deep inspiration through the slightly open mouth during the compression of the bag. Methods which do not require the act of swallowing have the advantage of causing less noise, and are therefore more favourable for auscultation; but the act of swallowing generally gives the most satisfactory results. Occasionally, through defective closure of the naso-pharynx, the air passes downwards and excites gastric pain, which is relieved by eructation or by a few full inspirations. Giddiness or tinnitus sometimes results, but these are temporary. If there is atrophy of the tympanic membrane, marked Eustachian patency, or acute tympanic inflammation, only slight pressure should be employed; otherwise rupture of the membrane or aggravation of the inflammation may result. In any case there should only be gentle pressure upon the bag at the first inflation. Whilst many patients are able to inform us correctly of the success of the inflation, the statements of others are not trustworthy, and they may say that they have no sensations in the ear when auscultation and inspection shew that the inflation has been quite successful, or vice versa.

By Valsalva's method the patient, while closing his nostrils with the fingers, makes a forced effort of nasal expiration. If successful he is usually conscious of a click and a sense of fulness in the ears; but many persons fail to inflate—even in a normal condition of the tubes. The effort is more likely to be successful if there be a perforation, and when the patient can inflate in this way there is not likely to be marked obstruction of the tubes. The frequent repetition of this method, it is to be remembered, is apt to be detrimental to the hearing. If a person swallow with the nostrils closed (negative Valsalva's method), some of the air in the middle ear passes into the naso-pharynx, and the membrane may be seen to recede. This produces a disagreeable sense of pressure, which is removed when the patient swallows with the nostrils open.

The information derived from these methods of inflation is obtained in several ways: (1) *Through observing the character of the sounds produced in the middle ear* when heard through an india-rubber tube of about 30 inches in length, furnished with an ear-piece at each end—the auscultation tube. This connects the auditory meatus of the patient with that of the surgeon. To ensure the proper conduction of sound nothing should be allowed to press upon or even touch the tube, and we must see that the ear-pieces are not obstructed with wax or other material. The student should always use the auscultation tube when inflating, so as to familiarise his ears with the various characters of the sounds heard. More information is gained in this way during catheterisation than by the other methods of inflation. In the normal state during catheterisation the sound has a distinctly blowing character, compared by Politzer to that produced when we place the tongue against the hard palate and make a quick expiration with the lips slightly apart. In undue patency, as in otosclerosis, the sound has a fuller, drier, and more frictional character than in the normal. When there is a thin fluid secretion in the tympanum or Eustachian tube, it has a moist crackling or bubbling character. A rough vibrating sound generally indicates that the point of the catheter is not properly inserted in the mouth of the Eustachian tube. If the sound be weak, somewhat distant, or interrupted, there is usually obstruction in the Eustachian tube or tympanic cavity; this is confirmed if it become fuller and stronger when the patient swallows at the moment of the inflation. When the tympanic membrane is indrawn but movable, the sound is usually very distinct, and of a full clicking or thudding character. A crepitating sound may be heard just after the inflation, due to the retraction of adhesions, etc., previously stretched. Probably the most characteristic sounds are heard in perforation of the membrane. If the perforation be small, with fluid secretion in the tympanum, there is a loud hissing sound, with perhaps gurgling. When the perforation is large a loud blowing sound is heard, sometimes painful to the listener, and conveying a sense of nearness; but, if there is narrowing of the tympanic end of the Eustachian tube, the sound may be of a whistling or squeaking character. Of course, when there is stenosis of the Eustachian tube these perforation sounds are not heard.

By Politzer's method auscultation does not yield such distinct information as is obtained by catheterisation, partly because the sound of the act of swallowing masks that in the ear, and also because in certain conditions no sound is produced by Politzer's method. The practised surgeon is, however, often able to associate many of the sounds, such as those connected with Eustachian obstruction, with the presence of secretion in the tympanum, with perforation, or with an indrawn movable membrane. The chief value of Valsalva's method is in helping to diagnose a perforation, when it is accompanied by the well-marked hissing sound. During this and the negative Valsalva method there is occasionally heard a slight clicking sound.

(2) *Inspection of the tympanic membrane,* during inflation, sometimes gives us more reliable information than auscultation. During Valsalva's experiment information may be obtained in this way, but much more so during Politzerisation and catheterisation. It is quite possible for the surgeon to carry out Politzerisation while inspecting the interior of the ear. In this way he may often determine the permeability of the tube and the mobility of the membrane by observing whether there is a bulging, or movement outwards, during the inflation. He may also determine the existence and site of a perforation by observing whether air bubbles, with secretion, appear at a particular place, or whether thick secretion is seen to protrude. He may sometimes see bubbles through the membrane after inflation, and this points to serous exudation in the tympanum. A change in the colour of the membrane may be noted as it moves outwards. A cicatrix or atrophied part usually bulges out like a bladder, disappearing shortly afterwards. Ocular demonstration of the effects of inflation may be given if a fine glass tube, having the shape of a horseshoe, is fitted air-tight into the external meatus, when the falling and rising of a coloured solution in the outer end of the tube will indicate the fluctuations of pressure in the middle ear and meatus.

(3) Important information regarding prognosis may be derived from observing *the effects of inflation upon defective hearing or sounds in the ear.* If, for example, in a case of chronic deafness, yielding little information by objective examination, effective inflation is followed by no improvement in the hearing and no mitigation of the tinnitus, the prognosis is unfavourable; there are probably sclerotic changes with fixations or stiffenings, or there is labyrinthine mischief. If, on the other hand, there is distinct improvement, with a feeling of relief, continuing for at least several hours, the prospect is favourable as indicating a more curable exudative middle-ear condition. If the improvement last for only two or three minutes there are probably adhesions, bands, or atrophy of the membrane—the temporary stretching during inflation causing a momentary improvement. In purulent perforative middle-ear disease, improvement in hearing and a sense of clearness after inflation, point to the likelihood that no serious defect of hearing will be left behind. Inflation is occasionally followed by a temporary feeling of increased deafness, probably from excessive tension of the tympanic membrane.

GENERAL SEMEIOLOGY OF THE EAR

The symptoms of ear disease may be conveniently described in the following order, the first four being much the most common:—(1) Defective hearing; (2) Sounds in the ear (tinnitus aurium); (3) Pain in the ear (otalgia); (4) Discharge from the ear (otorrhoea); (5) Vertigo; (6) Deaf-mutism; (7) Headache; (8) Psychical disturbances; (9) Intracranial symptoms; (10) Pyrexia; (11) Ocular disturbances; (12) Impairment of taste; (13) Facial paralysis.

(1) **Defective hearing** is the most frequent symptom of ear disease. It may vary from a defect so slight that the patient is unaware of its existence, to total loss of hearing. The latter, however, is very rare, even in deaf-mutes, of whom a large proportion hear intense sounds, such as a loud bell, near to the ear. In very many patients both ears are affected, although one is usually worse than the other. Patients not infrequently allege that they hear quite well, although the application of tests shews a real defect in one or both ears. Whilst defective hearing may be caused by pathological changes in any of the sections of the ear, the middle ear is the most common seat of the lesion; but the most profound forms are due to disease in the labyrinth or nerve.

It is well known that deaf persons often acquire great aptitude in reading the lips or facial movements, and in guessing what they do not hear from the sense of what they do hear. They are often surprised at the degree of their deafness, when tested with their eyes closed. Only by careful testing of the hearing, as already described (see p. 351), can we properly estimate the extent of the deafness. There is frequently a striking want of agreement between the hearing of a watch and that of speech. A patient may hear lightly spoken words fairly well, and yet not hear the watch, even when near to the ear; while, on the other hand, the hearing of words may be very defective and that of the tick of a watch comparatively good. Sometimes, indeed, the hearing of speech is better on the side on which the hearing of the watch is worse. During treatment we occasionally find that the hearing of speech distinctly improves, while that for the tick of a watch remains unchanged; or the contrary may be observed. Persons who have become deaf in later life usually hear speech better in proportion than the watch, while the opposite holds good with those who have become deaf in childhood. Probably the greater knowledge of language in elderly persons, as well as the unconscious power of reading the face, accounts for this better understanding of speech. There may be word-deafness without apparent ear disease. The power of forming word-pictures, apart from ordinary hearing, apparently resides in the cortex of the first convolution of the left temporal lobe.

Many persons, especially those over sixty years of age, have a defective capacity for hearing notes of a very high pitch; and this defect is also frequently noticed in persons, such as boiler-makers, who work amid noisy surroundings. In others we occasionally find that the ear interprets the tone incorrectly; instead of the real note from the sounding body, one is heard a half-tone, a complete tone, a third, or an octave higher or lower (paracusis). If only one ear be affected a double note is heard, namely, the true one on the normal side and the false one on the affected (diplacusis). Sometimes the tone or an echo of it is heard for a short period after the objective sound has ceased.

In certain forms of ear disease the patient hears better in a noise, for example when travelling in a railway carriage or in the presence of noisy machinery. This peculiarity is termed *paracusis Willisii*, because Willis first described a case in which a husband could only be heard by his deaf wife while the servant was beating a drum. It is generally considered to be due to the intense vibrations shaking the ossicles which have become stiffened; in this way it is thought they are made fitter for the transmission of ordinary sounds. Hearing better in a noise is, therefore, regarded as an indication of disease in the middle ear and not in the labyrinth.

Painful sensitiveness to sound, especially sounds of a very high pitch (hyperaesthesia acoustica), is apt to be experienced in fevers, in the early stage of middle-ear inflammation, in migraine, in persons who sleep lightly, or for a short time after the sudden removal of deafness. Even in persons who are very deaf, loud sounds, such as a railway whistle or speech through a conversation tube, may be very disagreeable or even painful.

There is sometimes inability to distinguish the direction of sound (paracusis loci), so that the report of a gun or the sound of a vehicle may seem to come from a direction opposite to the real source of the sound—a peculiarity which may obviously be fraught with danger to the person. This anomaly, when well-marked, is generally connected with unilateral deafness, since the power of localising sound is probably very largely due to binaural hearing.

In normal hearing, sound by air-conduction is more effective than by bone conduction; but, in persons with disease of the conducting structures, this is often reversed, and they may hear much better by bone conduction. Thus, the tones of a musical instrument may be appreciated by a very deaf person, if a rod of wood in contact at one end with some part of the instrument be held at the other end between the teeth of the patient. This also explains why the movements of mastication and the vibrations of his own voice (autophonia) may be heard much louder by a patient suffering from deafness due to disease of the conducting structures. The significance of increased and diminished bone conduction, in relation to air conduction, has already been considered on page 355.

(2) **Sounds in the Ear** (tinnitus aurium).—This is probably the next most frequent symptom of ear disease, and may be defined as sounds in the ear or head which have no objective cause outside the body. They are generally associated with defective hearing, and may be present in most of the diseases of the ear, although the worst forms are those due

to disease in the labyrinth, or in the nerve at its root or in its course. These sounds are extremely common, being probably present in about 60 per cent of cases of ear disease. They are frequently the symptom for which the patient comes for advice, as he is more anxious to be cured of the noises than of the defective hearing. Whilst the sounds are usually localised in the ear, they are described at other times as being in or through the head. They are in some cases really due to vibrations within the ear or in its neighbourhood (entotic and properly objective). For example, crackling or the sound of bubbles bursting may be due to secretion in the tympanum; a creaking or rubbing sensation may be due to cerumen or a foreign body in the external meatus; a slight drumming or buzzing may be caused by spasm of the tensor tympani or stapedius; while a crackling, heard on swallowing, is usually due to separation of the walls of the Eustachian tube when affected by catarrh. Again, a bruit may be heard in the jugular vein under the floor of the tympanum, while a pulsating or throbbing sound often arises from dilated arterioles due to congestion or vasomotor disturbance in the external or middle ear, or in the labyrinth. A pulsating sound may also be caused by an abnormal condition of the internal carotid artery in the carotid canal; in this case the pulsation is usually stopped by pressure upon the artery in the neck.

Probably, however, the worst forms of tinnitus are the purely subjective, caused by irritation of the auditory nerve, either due to pressure upon the roots or trunk of the nerve in the cranium, or to a primary lesion in the labyrinth acting upon the terminals of the cochlear nerve, or to intra-labyrinthine pressure through the fenestrae induced by middle-ear disease. The irritation may also be due to vasomotor changes in the labyrinth, reflex in origin, arising from more or less distant regions, such as the intranasal spaces or the digestive organs. The character of these purely subjective sounds is extremely varied. They are frequently described as buzzing (or "bizzing"), hissing, singing, ringing of bells, whistling, humming, or the sound of a sea shell. Those of an intense character are compared to steam blowing off, the noise of machinery, the rushing of a waterfall, the constant whistling of a locomotive, or the sound of a horn. More rarely musical sounds, even complete pieces of music, are heard. Several different sounds may exist at the same time, such as a pulsating and a buzzing. Tinnitus like the ringing of bells, the running of water, or music, may at first be confounded with real sounds, and may seem so genuine as to compel the patient to search for their source outside.

Whilst tinnitus may give but little annoyance, on many persons it has a most worrying and depressing effect, hindering work, preventing social enjoyment, and apparently rendering life a burden; indeed, cases are on record of patients seeking oblivion in self-destruction. Usually, during stillness and solitude, the noises are more prominent and disturbing. They are apt also to be aggravated by alcoholic stimulants, by over-exertion, by mental emotion, by worry and nervous irritation; they are also often said by the patients to be worse during colds in the head or in dull, damp weather. While they probably occur more readily in neurotic persons, a permanently nervous condition may be excited by them. In many the sounds are intermittent, in others they are unceasing, although they seem only exceptionally to prevent sleep.

Tinnitus may be present when there is but slight impairment of hearing, and occasionally it is met with in persons with normal hearing; but in such cases, if persistent, defective hearing frequently comes on sooner or later. More commonly the sounds are accompanied by marked deafness, and the patient may be deaf to all external sounds, while tormented by noises in his ear or head.

Tinnitus is sometimes associated with hallucinations of hearing in the insane, and cases are on record of the cure of melancholia with hallucinations of hearing by the removal of impacted cerumen. The continuous hissing, buzzing, etc., attending an ear disease may be associated with the hearing of "voices," which have been known to disappear under the local treatment of the ear disease.

(3) **Pain in the Ear** (otalgia).—This is also a frequent symptom; few persons pass through childhood without an attack of earache. Pain in the ear may be inflammatory or non-inflammatory. The former is much the more common, and tympanic inflammation is the cause of the most frequent and severe pain (otitis media acuta). In this case there is first a sense of heat, fulness or pressure, going on to dull pain, or, more commonly, to sharp, penetrating pain often extending to the temple above, the occiput behind, or the forehead in front, and frequently attended by throbbing. The pain is usually aggravated at night. In the purulent form the pain, after from an hour or two to a week or two, ceases with rupture of the membrane and discharge from the meatus; the pain, however, sometimes continues after rupture, or may return after a period of intermission. In connexion with tympanic inflammation, pain in the mastoid region is common, especially severe and increased by pressure in periostitis with oedematous swelling or subperiosteal abscess. When the mastoid cells are affected, but not the periosteum, the pain may be but slight and mainly elicited by pressure upon the front of the lower half of the mastoid. Pain on pressure behind the mastoid may denote mastoid empyema or implication of the sinus. Tenderness on pressure over the mastoid glands must not be confounded with periostitis. Pain behind the angle of the jaw may be due to glandular mischief in connexion with a purulent middle-ear disease, or it may point to septic thrombosis of the internal jugular; careful examination along the course of the vein is necessary. Pain in front of the ear may indicate extension of the inflammation to the parotid gland. In most cases of chronic purulent otitis media, pain is absent for years; on the other hand, severe pain may arise from an acute exacerbation, (i.) due to retention of inflammatory products set up by stenosis of the meatus or by other means; (ii.), from caries or necrosis; or (iii.), perhaps most frequently, from furunculi in the meatus. It is to be

remembered that pain, beginning in an ear affected with chronic purulent disease, and extending to the side or back of the head, may be the first symptom of an intracranial complication. Slighter or more intermittent pain may be complained of in simple middle-ear catarrh and even in the course of sclerotic processes; when severe, it may mean an intercurrent acute inflammation. The external meatus is a common seat of pain, most frequently due to furunculi in its lining, when it is aggravated by moving the auricle, by pressing the tragus, or by movements of the jaw as in mastication. This, as a source of pain, is often overlooked, and careful examination of the skin of the meatus with the help of a probe is necessary. Less frequently pain in the meatus is due to eczema, to fungi, to pressure of cerumen, to a foreign body, or to caries or necrosis. In chronic eczema the sensation is more that of itchiness.

Non-inflammatory or neuralgic pain is often dental in origin, especially from the molars of the lower jaw, and associated with pains in the neck, temple, or cheek. Severe reflex earache is sometimes experienced during a tonsillitis, also in carcinoma of the tongue or throat, and more rarely in disease of the accessory sinuses of the nose. Constitutional disturbance, such as anaemia, may be the cause of the earache. A sensation as if a plug were in the ear is sometimes complained of by persons with good hearing, and is evidently neurotic in character. Pain in the articulation of the lower jaw in front of the ear, felt during movement or pressure, and usually rheumatic in nature, is sometimes thought by patients to be due to ear disease. Examination with the mirror and speculum is the only trustworthy means of distinguishing an inflammatory from a neuralgic pain; and this should never be omitted, where pain in the ear is complained of. We often find that the neuralgic pain has been experienced intermittently for a long period, even for months, without deafness or any other symptom of ear disease, while, in the inflammatory form, the pain is usually recent, and there is more or less defect in hearing, generally along with subjective sounds. It is well to inquire whether there have been earaches in childhood or later on, as these may indicate past inflammatory attacks.

(4) **Discharge from the Ear** (otorrhoea).—Probably about a third of all diseases of the ear are attended by discharge which, with comparatively few exceptions, has its source in the middle ear, and is due to purulent otitis media, with perforation of the tympanic membrane. In a certain limited number a discharge may be due to inflammation of the external meatus, especially from eczema. While pain is frequently present at the early stage it is often absent, but there is generally more or less defective hearing. The discharge has, in many cases, existed for months or even years before the patient comes under the surgeon's notice. It may be very slight in quantity, so as even to escape the attention of the patient or be mistaken by him for "thin wax," or, on the other hand, its quantity may be such as to fill the meatus very soon after the ear has been syringed. The discharge is most commonly muco-purulent, but is often serous in the early stage of the inflammation, or when due to

eczema of the meatus. When the mucous element predominates it has a tenacious or stringy character, while, if purulent, a milkiness is imparted to the liquid escaping from the ear on syringing. The discharge is sometimes tinged with blood, especially if there are granulations or polypi in the ear, and has frequently an offensive smell, especially in neglected cases; in some cases the odour resembles that of old cheese. In all cases of otorrhoea, objective examination is essential; and, in order to see the parts properly, it is in most cases necessary to syringe the ear and dry the interior before using the mirror and speculum. Epithelial debris may form an obstruction to the view somewhat difficult to remove. Where there is a history of past discharge, we often find a dry perforation in the tympanic membrane, a cicatrix, or calcareous deposit.

In order to determine the greater or less gravity of the disease, it is very desirable that the discharge from the ear should be bacteriologically and cytologically examined. Bacteriologically the most serious forms are associated with the diplococcus and the *Streptococcus pyogenes*. The former includes the *Meningococcus* (Weichselbaum), the *Gonococcus*, *Micrococcus catarrhalis*, and the Gram-positive pneumococcus. The *Staphylococcus pyogenes albus* and *aureus* are much less virulent than the others, and are found in connexion with furunculi in the meatus as well as in purulent middle-ear disease. The tubercle bacillus is often found in children. Cytological examination, which should be repeated in any case more than once, yields, in the opinion of so high an authority as Dr. Milligan, very important information. His conclusions are that the presence in the discharge of lymphocytes indicates granulation-tissue formation; of lymphocytes, epithelioid cells, and myelocytes, bone disease; of lymphocytes and "acid-fast" squames, cholesteatomatous changes; of lymphocytes, epithelioid cells, myelocytes, and giant cells, tuberculous disease of the temporal bone. The complications and dangers associated with discharge from the ear are described elsewhere (see p. 475).

(5) **Vertigo.**—This, as a sign of ear disease, is far from being rare; In 6 per cent of my cases I found that it was a distinct feature. It manifests itself in various forms, the most striking is connected with labyrinthine or nerve disease, of which the most typical is true Menière's disease (sometimes termed apoplectiform deafness), due usually to haemorrhage or exudation into the labyrinth. In this form of aural vertigo a person, previously with normal hearing, is suddenly seized with vertigo, tinnitus, and deafness in one or both ears, quickly followed by sickness and vomiting; there are also pallor of the face, cold sweats, and in some cases fainting. The sickness and vomiting usually begin soon after the giddiness, and are probably due to the close relation between the nucleus of the auditory nerve and that of the vagus. The sickness and vomiting may suggest brain mischief; on the other hand, they are often regarded simply as indications of a purely bilious attack. Another form of aural giddiness, with, it may be, the whole series of Menière's symptoms, is due to sudden or perhaps gradual pressure upon the walls of the middle ear exerted by air, as in Valsalva's experiment,

by liquid, as in syringing when there is a perforation, or by inflammatory products, such as pus or cholesteatomatous masses. Vertigo may even be due to pressure upon the walls of the external meatus or upon the tympanic membrane, for example, by impacted cerumen. In a third class the vertigo is connected with chronic processes in the middle ear, such as sclerosis, "dry" catarrh, or post-purulent changes, probably, however, affecting the labyrinthine fluid by pressure through the fenestral openings or by exciting vasomotor disturbance in the labyrinth. This class answers to the *Vertigo ab aure laesa* of writers; sickness and vomiting being often absent, whilst recurrences of the giddiness are common.

When vertigo is due to extension of a purulent ear-disease to the cranial cavity, especially the cerebellum, it is associated with grave symptoms, pointing more to central structures than to the organ of hearing. It is to be noted, however, that vertigo may be the first symptom of cerebellar tumour independent of the ear. It must not be forgotten that Menière's series of symptoms often exists in connexion with purulent ear-disease which has not extended beyond the cavities of the ear.

In aural vertigo, the giddiness may be so extreme that, if the person cannot clutch a firm support or be supported, he falls to the ground or has to lie down; or it may amount to a mere sense of "swimming in the head," when turning quickly or stooping, or to a tendency to reel when trying to walk in a straight line. The giddiness may be experienced in bed, the patient feeling as if the bedroom were turning round, or the bed rising or sinking. In the severe forms the objects around may appear to rotate, or the ground in front may seem to rise or fall, or there may be a feeling as if he were on a suspension bridge which sways under the feet. The sense of movement may be confined to the patient's body, perhaps of the nature of a rotation round a vertical axis, when the tendency usually is to turn or fall towards the affected ear, or there may be a sense of moving backwards or forwards on a horizontal axis. Experiments on animals seem to indicate that irritation in the external semicircular canal causes horizontal movements of the head, whilst irritation in the posterior or superior is attended by movements backwards and forwards. In other cases there may simply be a staggering or want of control of the legs, especially in the dusk or dark, the movements resembling those of a drunken man. The giddiness may pass off or markedly diminish in a few minutes, or it may last for several days, necessitating the lying posture. It may return, even repeatedly, with more or less severity. A certain degree of giddiness or tendency to reel may persist, with exacerbations from time to time, perhaps excited by other causes, such as gastric disturbance. Whilst, in aural vertigo, there is good reason to believe that irritation of the vestibular and ampullary nerves, sending afferent impulses to the centre of equilibrium, is the real source of the mischief, there may supervene other exciting causes which provoke attacks, such as digestive or hepatic disturbance, nervous shock or strain, exhaustion.

Without these, although the ear disturbance may be permanent, the vertigo may be absent for long periods of time.

(6) **Deaf-Mutism** is in the vast majority of cases simply a consequence of a high degree of deafness, either congenital or originating in early life (acquired). Probably 75 per cent of deaf-mutes hear such sounds as a loud bell or a whistle close to the ear, or a vowel strongly pronounced into the ear. In a smaller number, complete words are heard when spoken loudly into the ear or through a tube. It is not easy to determine positively, in the first year of life, whether a child hears. The history of a case is generally that the mother is surprised to observe that the child is unaffected by noises which awake other children. After the first year, her anxiety may be aroused by the delay in beginning to speak, and by manifest inattention to loud sounds. Suspicion being excited, the child's hearing should be tested, preferably by the parents at home under the directions of the doctor (see p. 354). By objective examination, morbid changes may be seen in the tympanic membrane; but, in most cases, these do not account for the serious deafness, the cause of which is more deeply seated.

In less severe forms of deafness, though mutism may not result, there is apt to be a less perfect articulation and a lack of correct intonation. If the deafness become worse, although not total, the speech is apt to become more and more imperfect, and the child's knowledge and memory of words being insufficient to enable him to retain what he has acquired, he may cease altogether to speak or simply make unintelligible sounds. After a time the parents, believing that it is useless to speak to the child at all, resort more and more to gestures, and he comes to be classed as a confirmed deaf-mute.

(7) **Headache** is not infrequently connected with ear disease. In acute otitis media, shooting pains are often complained of in the temple and occiput, and these are frequently regarded as neuralgic, because, in the absence of objective examination of the ear, the real cause has been overlooked. In the chronic cases similar but less severe pains may be felt; and, in purulent disease, there is often a heavy dull pain in the head. A benumbed sensation over the corresponding side of the head is common in acute middle-ear catarrh or inflammation, whilst in the chronic forms, there is often a sense of pressure on the top of the head. When, in chronic purulent otitis media, pain in the head is sudden, severe, and continuous, especially if attended by sickness and vomiting, we should think of the possibility of such a complication as abscess of the brain, meningitis, or thrombosis of the lateral sinus. Mastoid inflammations are also usually attended by pain in the neighbouring parts of the head.

(8) **Psychical Disturbances.**—As might be expected, school children with defective hearing are usually more backward than hearing children, and there is no doubt that, by the marked impairment of hearing in youth, the mental faculties are likely to suffer, and the child is apt to become inaccurate in statement and undecided and unstable in disposition.

A special form of diminished power of mental application with loss of memory, termed by Guye *aprosexia*, is frequently noticed in children affected by post-nasal growths and middle-ear catarrh. Deaf adults, especially when suffering from tinnitus, often complain keenly of a feeling of heaviness, confusion, or depression, probably due to the distracting influences of noises in the ear as well as to the strain of attempting to hear.

(9) **Intracranial symptoms,** such as delirium, convulsions, stupor, coma, and paralysis, are not infrequently met with in connexion with purulent middle-ear disease. In the presence of such symptoms, the existence of otorrhoea is of great significance and imperatively calls for careful examination of the ear.

(10) **Pyrexia.**—Some fever is found in most cases of acute otitis media, and even of otitis externa. In some cases the rise of temperature is very considerable, not infrequently reaching 102° F., especially in children. Still higher temperatures are observed in acute mastoid inflammations. When such complications as meningitis or septic thrombosis supervene, the fever becomes a very notable feature; and in the latter complication it presents striking intermissions, each rise of temperature being ushered in by a severe rigor.

(11) **Ocular disturbances** in the form of iritis or keratitis or both, are often associated with hereditary syphilis affecting the labyrinth. In purulent ear-disease, changes in the fundus of the eye are not infrequently noticed, especially if associated with cranial or vascular complications, when distinct optic neuritis is common. It has been shewn that vascular changes in the optic disc, short of optic neuritis, are very common in persons with purulent ear-disease even when there is no evidence of intracranial complications. Paralysis of one or more of the ocular muscles is a well-known symptom of the intracranial complications of ear-disease, especially in temporo-sphenoidal abscess when the third cranial nerve is frequently involved. It has been demonstrated, both by experiments and by clinical observation, that nystagmus may arise reflexly from labyrinthine pressure or irritation, especially in the semicircular canals, when it is generally associated with pronounced giddiness. Nystagmus is observed most frequently in purulent ear-disease, especially in labyrinthine suppurations, and it is often present in cerebellar abscess. Temporary attacks may be induced by syringing the ear, or by manipulating the deep parts, such as applying the probe, or removing granulation tissue or a polypus.

(12) **Impairment of the sense of taste,** especially on one side of the tongue, corresponding with the side on which a purulent ear-disease exists, may sometimes be found, if careful testing be carried out. It is caused by implication of the chorda tympani nerve in the tympanic cavity. Pressure upon this nerve, when the upper part of the tympanic membrane is destroyed, excites a peculiar taste on the corresponding side of the tongue. A disagreeable taste is sometimes complained of, apparently due to the escape of pus from the Eustachian tube into the pharynx.

(13) **Facial paralysis** is not an infrequent symptom of ear disease,

especially of the purulent forms. Occasionally it results from simple middle-ear catarrh, and has been found associated with herpes of the auricle or meatus. It is generally unilateral, affecting the side corresponding with the ear disease. If due to an intracranial lesion, it is found on the opposite side, and it is then more partial in character. Rarely the paralysis is bilateral, and, in the only case seen by me, it was due to syphilitic disease, implicating also the auditory nerves. Facial paralysis may also be the result of the radical mastoid operation. The symptoms produced by pronounced facial paralysis are well known, being chiefly due to the inaction of the facial muscles on the affected side. A minor degree is probably more common in connexion with purulent ear-disease than is supposed, shewing itself mainly in a less distinct naso-labial furrow. Different fibres of the nerve, having different areas of distribution, may be more markedly affected than others. The brow and the eyelids may, for example, be more affected than the mouth or cheek, or vice versa. When the lesion is above the origin of the twig for the stapedius muscle, there may be disturbances of the hearing with tinnitus, caused by inaction of the stapedius muscle.

Thomas Barr.

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GENERAL THERAPEUTICS

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In many cases of ear disease local treatment will be more efficacious if the general health be good, and hence constitutional treatment also is often necessary. As will be seen later, tuberculosis, syphilis, and anaemia among other conditions may dispose to ear disease, and these etiological factors must not be lost sight of in treatment. Morbid conditions in the naso-pharynx also commonly cause aural disease, and must be remedied. Climate, too, exerts some influence; thus, dry mountain air is particularly suited for cases of chronic non-suppurative inflammations of the middle ear, whilst the seaside, prejudicial to such conditions, is frequently beneficial in chronic suppuration. Without elaborating these statements further, we may at once turn to the consideration of local measures.

Syringing the Ear.—The syringe should be one that can be sterilised by boiling, and should be furnished with a nozzle sufficiently fine to enter the external meatus. The liquid employed should be sterile saline solution or boracic lotion 1 in 40; ordinary boiled water should not be used, as it is apt to irritate the mucosa of the middle ear, if there be a perforation in the tympanic membrane. The lotion should always be warmed before use, as when cold it is much more apt to produce unpleasant symptoms; the exact degree of warmth is immaterial, anything up to 98° F. will do. After filling the syringe bubbles of air must be removed; this is easily managed by directing the nozzle of the instrument upwards and pressing the piston in until all the air has been expelled. The patient should be seated, as giddiness or even syncope may supervene; a towel is laid over the shoulder and the patient holds a receptacle—a china bowl is sufficient —under the ear and in close contact with the skin in order to catch the lotion. The parts are well illuminated either by direct or by reflected light. The auricle is pulled upwards and backwards in an adult—downwards and backwards in an infant, who should be held in an attendant's arms with the head steadied, and the fluid is then injected along either the upper or the lower wall of the meatus. Excessive force must not be exerted. In attempting to remove hard wax or plugs of epithelium which will not come away easily, it is better to soften the mass first by the instillation of a solution of bicarbonate of sodium in glycerin. Frequently, however, the desired result may be obtained by directing the patient to open the mouth widely while the ear is being syringed. It is advisable in any case not to continue the syringing if pain be induced, lest inflammation of the external or middle ear be set up.

It is sometimes desirable to syringe the middle ear directly, for instance, in attic suppuration or in cases with a perforation extending to the annulus tympanicus. This form of treatment was first put into practice by Schwartze, though it is usually associated with Hartmann's name. Hartmann's instrument consists of a fine cannula bent up at the one extremity, and with a piece of rubber tubing terminating in a small rubber ball attached to the other end. The instrument is sterilised and filled with warm sterile saline solution, and any adventitious air is expelled from it. The surgeon sits opposite the ear to be syringed, and using a forehead mirror and speculum, holds the instrument in his right hand between the index finger and thumb, and grasps the rubber ball in the same hand. The point of the cannula is directed through the perforation, and the hand is then steadied by placing the little finger against the patient's cheek; the lotion is then slowly pressed from the ball. The syringing may be repeated several times at each sitting; for the last washing rectified spirit may be employed to assist in drying out the middle-ear cavities. Finally, the air douche must be used, partly to expel the lotion remaining in the tympanum, and partly to obviate the giddiness which is nearly always produced to a greater or less extent. It may be necessary to continue this method of treatment for weeks, and it should, if possible, be carried out daily, so that its application is rather limited. I have, however, seen more than one case of cholesteatoma apparently cured by this means alone. After syringing the ear by either of these methods, the meatus must be carefully dried by passing a probe, roughened at the end and dressed with cotton-wool, down to the tympanic membrane. When syringing is carried out at home a too-powerful instrument should not be ordered, a glass or Higginson's syringe with a suitable nozzle may be advised. Those responsible for its performance must be told to observe the greatest cleanliness in carrying out the treatment. The syringe must be frequently boiled or washed out with carbolic lotion, the receptacle for the lotion should be scalded, boiled water only should be used, and the hands must be well scrubbed. It is desirable that the drying out of the ear should be left entirely to the patient, a match may be used instead of a probe, the non-luminous end is dipped in the lotion and dressed with cotton-wool, the end being moistened in order that it may adhere the better; it is then passed deeply into the meatus, the auricle being pulled upwards and backwards by the other hand. The wool, which is sterilised, is stored in a glass jar, enough being removed for immediate use at each sitting by a pair of forceps, the ends of which have first been passed through a flame. After the ear has been dried it is as well to insert a plug of wool into the meatus to guard against any irritation.

Occasionally small quantities of fluid are injected into the middle ear through the Eustachian tube in cases of non-suppurative inflammation, some drug, such as iodide of potassium, bicarbonate of sodium, or menthol being dissolved in the fluid—oil being used as the solvent for the last, in the hope, to my mind a vain one, that the hyperplastic tissue will thereby be caused to disappear. The procedure is carried out in the following way: a catheter with a well-marked curve and a wide lumen is chosen. This is passed in the usual way, and the surgeon satisfies himself by inflation and auscultation that the beak is in the mouth of the Eustachian tube. He then injects a few drops of the solution into the catheter by means of a small syringe, provided with a nozzle which accurately fits the bell-mouth of the catheter, and immediately forces the fluid through the Eustachian tube into the middle ear by means of Politzer's bag. Larger quantities of fluid are occasionally injected by this route in order to wash out the tympanum in chronic suppuration.

Introduction of Vapours into the Middle Ear.—Vapours are sometimes introduced into the middle ear. The simplest method of doing this when the Eustachian tubes are patent is to fill the mouth with the fumes and then to perform Valsalva's experiment (*vide* p. 367). In this way chloride of ammonium and iodine inhalations may be used. Another plan is to insert the nozzle of a compressed Politzer's bag into the mouth of a flask containing some volatile substance, such as menthol or turpentine, and allow it slowly to expand; the bag will fill with the vapour, which is then driven through a catheter into the middle ear.

Introduction of Medicated Solutions into the Meatus.—Instillations of medicated solutions into the meatus are prescribed frequently, and certain instructions must he given to those entrusted with the treatment. Among the remedies most commonly employed are carbolised glycerin, rectified spirit, peroxide of hydrogen in solution, and menthol in paroleine. All these solutions, with the exception of the rectified spirit, should be slightly warmed before use. The patient bends his head to one side, the affected ear being uppermost. The auricle is pulled upwards and backwards and about ten drops are then poured into the meatus, the tragus is pressed on immediately afterwards in order to drive the fluid inwards. If there be a perforation of any size in the drum membrane, some of the solution may find its way into the throat. When only a temporary action is required the ear should be carefully dried out, as already described, after the lapse of some five minutes; but if more prolonged action is necessary a pledget of wool is inserted into the meatus, the excess of fluid merely being first allowed to escape.

Insufflation of Powders.—Though some cases of otorrhoea may be greatly benefited by powders, stress must be laid on the danger that attaches to their use in cases in which there is any risk of interfering with the drainage. Powders, therefore (as M'Bride and Schwartze have pointed out), should never be used in a chronic case with a small perforation. The method of employment is very simple: after the ear has been syringed and dried out the powder is blown in by an insufflator with a fine straight nozzle; a sufficient quantity should be used to form merely a thin film. Some authorities who prefer the so-called dry method merely mop away the discharge before introducing the powder.

Caustics are of value in burning small granulations or in destroying the stump of a polypus which has been removed by the snare; of the various forms at our disposal, apart from the electric cautery which will be described later, solid nitrate of silver or chromic acid are the most efficacious, and should be applied fused on a probe. When dealing with a nervous patient a few drops of cocaine may with advantage be instilled a few minutes before the caustic is applied. The ear should be well illuminated, and the surgeon making use of a forehead mirror and speculum takes care only to touch the part on which it is desired that the caustic should act. If too wide an area be burnt the action can be arrested at once by the instillation of a solution of chloride of sodium when nitrate of silver has been used, and bicarbonate of sodium in the case of chromic acid. The introduction of a solution of nitrate of silver has been recommended in certain cases of chronic otorrhoea, but considering the difficulty of limiting the action of the caustic if used in this way, the method would appear not altogether advisable.

Electricity has been used in various forms in the treatment of ear disease. The interrupted current has occasionally been of service in cases of tinnitus, one pole being applied over each mastoid process, while the current is allowed to pass for a few minutes. The continuous current has also been employed, but its value is very doubtful. A simple method is to place the positive electrode against the tragus while the negative is held in the hand. The current is then closed. This form of treatment may be used in cases of nerve deafness, and is based on the results of Brenner's experiments, which shewed that when the current was made with the poles arranged in the opposite way, a sensation of sound was caused which ceased on breaking the current. The electric cautery may be employed to make perforations in the drum membrane, which it is desired should remain open for some time. It may also be used to burn away granulations. A very fine burner must be chosen, and a solution of cocaine should be previously instilled into the ear.

The value of electrolysis in the treatment of so-called stenosis of the Eustachian tube has been very highly appraised, especially in America. The method, chiefly associated with the name of Duel of New York, is carried out in the following manner. The mouth of the Eustachian tube is cocainised, and an ordinary silver Eustachian catheter, wound with rubber tissue for the purpose of insulation, is passed in the usual way. A bougie of gold wire, sufficiently long to project 1¾ inches beyond the beak of the catheter, and with an olive tip 1 to 2 mm. in diameter, is passed through the catheter into the Eustachian tube until it meets the obstruction. The wire bougie is made the negative electrode, and the positive is the ordinary contact electrode held in the hand of the patient. A current of 1 to 5 milliampères is turned on, while the tip of the bougie is held firmly against the obstruction,—the bougie is said usually to pass through the stricture in a fraction of a minute. It may be necessary to repeat the process several times before the instrument enters the tympanum. It is stated that re-absorption of inflammatory connective tissue takes place. It is, however, extremely doubtful whether stenosis of the Eustachian tube, the pathological condition for which this method of treatment has been devised, exists; Bezold has not met with a single case in hundreds of autopsies; Duel's observations must therefore be regarded with some scepticism.

Anaesthesia in Aural Operations.—For major operations a general anaesthetic is required, whilst for minor operations local anaesthesia is usually sufficient except in nervous children. A solution of cocaine hydrochloride alone has but little anaesthetic action when instilled into an ear with an intact membrane, but if the drug be dissolved in aniline oil and spirit, as first suggested by Dr. Gray, a considerable degree of anaesthesia may be obtained, the reason being that the aniline oil penetrates the tissues and brings the cocaine into direct contact with the nerve endings, while the spirit aids by absorbing any moisture that may be present. There is an objection to the solution—hypothetical rather than practical—in that the aniline oil itself is toxic, and therefore an excessive amount of the solution should not be instilled. As regards its strength, 5 to 10 parts of cocaine hydrochloride to 50 parts each of spirit and aniline oil will be found a useful formula. It certainly has the disadvantages that it does not obviate what is frequently a serious difficulty, namely, the haemorrhage, and that it has not any marked action on a highly inflamed drum. Bonain's formula, which is efficacious in acute inflammatory conditions and also in other cases requiring local anaesthesia, is as follows: carbolic acid, menthol, cocaine hydrochloride, of each 1 grm., and adrenalin chloride 1 mgrm. It is applied by means of a piece of cotton-wool soaked in the mixture and left against the tympanic membrane for from three to five minutes. In those cases, however, in which more has to be done than mere incision of the drum membrane, it is more satisfactory to inject a solution of cocaine hydrochloride and adrenalin chloride after Neumann's method. The syringe is made entirely of metal, and the barrel is furnished with half rings, into which the fingers are inserted when pressing the piston home. The nozzle beyond the cylinder is bent at an angle common to all aural instruments, and ends in a screw-thread on to which the needle itself is screwed. The needle is considerably stronger than that of the ordinary hypodermic syringe, and is about two inches long. When giving an injection, the syringe is filled with a 0·5 per cent solution of cocaine hydrochloride containing two or three drops of a solution of adrenalin chloride (1 in 1000). The needle is introduced through a speculum into the external auditory meatus, the point is thrust below the skin at the upper and back wall of the meatus about 1 cm. from the drum membrane, and the piston is then slowly pressed in. If the injection be successful the parts will at once blanche, and if there be a perforation in the membrana tympani, drops of the solution will escape through it from within the middle ear. The patient must be prepared to find the prick of the needle somewhat painful. After waiting a few minutes the surgeon will be able to begin, and if he has been successful there will be no bleeding and no pain. Some practice is required in making a successful injection. The procedure has been further elaborated, and Neumann and Alexander state that they have performed radical operations painlessly by this method. In such cases a solution of cocaine hydrochloride is also injected below the periosteum over the mastoid process.

Operations.—Though a detailed description of the various operations performed on the ear is unnecessary here, some general statements must be made as to the technique and choice of instruments. Most instruments constructed for use in the external auditory meatus are bent at an angle of rather over 90°, with the object of bringing the hand below the axis of vision when using the instrument. It is customary to have a set of knives, curettes, blunt and sharp hooks, and the like, which can be fixed in a common handle; the latter should be roughened and not too delicately wrought. A useful form of knife for incising the membrane is one with a small diamond-shaped blade, such as Politzer's. For excision of portions of the membrane, however, a sharp-pointed knife with a narrow blade is to be preferred. For incision of furuncles in the meatus a tenotomy knife is as suitable as any specially designed for the purpose.

Various forms of aural snare have been invented. The best and simplest is probably the original Wilde's snare, or one of its modifications, and it is advisable to select one with the extremity delicately wrought so as to avoid obstructing the view. The snare is armed with a fine iron or copper wire. When dealing with polypi too small to be caught with a snare, a ring-knife or a curette may be used. The inner edge of the blade of the ring-knife is sharp, and the ring is inserted so as to press against the granulation, which is then scooped out. Growths with a comparatively wide base can be removed satisfactorily in this way, or by small curettes or sharp spoons. The double curettes made for the ear have little practical value, and are certainly not worth the high price charged for them. Small hooks, sharp or blunt, are occasionally useful in extracting foreign bodies.

During the routine examination of the ear a pair of forceps, such as Politzer's angled forceps, should be at hand to remove shreds of epithelium, flakes of wax, and the like; the inner aspects of the points should be ribbed to prevent the blades from slipping. Two kinds of probes are required: one slender and blunt-pointed, to be used as an aid to diagnosis, the other stouter and having a screw-thread on the end which can be readily dressed with a piece of wool. With such a dressed probe any secretion in the meatus can, in my opinion, be more easily and quickly mopped out with a view to inspection of the membrane than by introducing a wick of wool down the meatus by means of angled forceps.

The specialist will also provide himself with instruments for dividing the tendon of the tensor tympani muscle, and for extraction of the ossicles. For the former purpose I prefer Schwartze's tenotome, which has a blade curved on the flat; whilst for removal of the hammer an ordinary snare will be found useful or Delstanche's special form of ring-knife, and for dislodgment of the incus some form of hook, such as Ludewig's is required. Gouges for the major operations on the mastoid process should be made shallow, and the cutting edge should on no account be pointed, but should lie in a plane at right angles to the shaft; the corners should be slightly bevelled. With such an instrument the surgeon may boldly remove large chips of bone without fear of wounding the sinus, even when situated unusually far forward, for if exposed it will be pushed back and not injured.

Rarefaction and Condensation of Air in the External Ear.—Rarefaction of air in the external ear can be simply produced by introducing into the meatus an accurately fitting ear-piece attached by a short piece of rubber tubing to the nozzle of a Politzer's bag. After adjusting the ear-piece the bag, which has been previously compressed, is allowed to expand, and in this way suction is applied to the drum membrane. It is obvious that the force cannot be properly regulated by this method, and accidents have occurred, such as extravasations of blood within the membrane or tearing of that structure. It is, therefore, wiser to use an instrument specially devised for the purpose, by which the amount of suction can be varied as desired; of these, Delstanche's raréfacteur or that designed by Cordes are suitable.

It may be desirable to produce an alternating rarefaction and condensation of the air in the meatus, the phases being made to succeed each other at a greater or lesser speed. The simplest method of procedure is, as was suggested by Hommel, to get the patient to press with the finger on the tragus and immediately to release the pressure. The process may be repeated as rapidly as possible for a minute or so, and should be carried out three or four times a day; or the surgeon can induce fairly rapid alternations of pressure by first squeezing and then relaxing the ball attached to a Siegle's speculum, which has been adjusted within the meatus. At the same time he can observe the effect on the drum membrane. Advocates of this principle of treatment will, however, probably prefer to use more elaborate instruments, such as are capable of producing very much more rapid alternations of pressure. For this purpose air-pumps may be obtained, an ear-piece being connected by rubber tubing to the air-pump, while the pump itself may be driven by hand or by an electric motor. The method is known as pneumomassage.

Inflation of the Middle Ear.—The various means of inflating the middle ear have been described on p. 362, but it is necessary to indicate here the therapeutic value of this form of treatment, and to consider the respective advantages of the different methods. Whatever means be employed, the chief object is to make the pressure of air within the tympanum equal to that of the atmosphere; in other words, to ventilate the middle ear. A second important indication is to dispel collections of fluid within the tympanum, for, as will be readily understood, the sudden inrush of air will drive the liquid into the meatus, if there be a perforation in the drum membrane, whilst if that structure be entire the secretion will be scattered through the middle-ear cavities, and will then be more readily absorbed. A third important indication is to prevent the formation of adhesions within the tympanum, or when adhesions exist to rupture or stretch them.

As regards the choice of method, the carrying out of Valsalva's experiment is usually quite inadequate when inflation of the middle ear has to be systematically performed as part of the treatment. The surgeon is, therefore, in practice restricted to Politzer's method with its modifications, or to the use of the Eustachian catheter. The technique of the former is of course the easier, and this method is the only one applicable in the treatment of children, and being so simple, it may be relegated to the friends of the patient. The disadvantages are that it is impossible to restrict the action to one side, and therefore when one ear only is affected there is a risk of causing undue relaxation of the drum membrane on the healthy side. This danger must be remembered, and the aurist should satisfy himself by inspection of the membrane at short intervals that all is in order. Finally, there is a greater risk that a cicatrised or calcareous membrane may be burst, as the force is less easily regulated than when using a catheter. The disadvantages of employing the catheter are the difficulty in passing it, and the discomfort that it causes the patient; but the great advantages are that the action can be confined to one ear, that the auscultatory phenomena can be readily observed, and that the force of inflation can be regulated.

Lucae's pressure probe, as a method of treatment, was introduced by the Berlin School, but has not found much favour elsewhere. The instrument consists of a probe which rides in a hollow handle, and is attached to the base of the socket by a fine steel spring, thus enabling varying degrees of pressure to be applied. When it is desired to use the instrument, the end of the probe, protected by a pledget of cotton-wool, is placed against the short process of the malleus, and slight pressure is then exerted and immediately relaxed. This alternating pressure and relaxation is quickly repeated for from four to thirty times, according to the tolerance of the patient, who should be warned that when the probe is placed in position a certain amount of pain will be felt. The treatment is chiefly restricted to cases of non-suppurative catarrh of the middle ear. Lucae has more recently designed an electro-pneumatic pressure probe: in the handle of this instrument there is a thin membrane connected by rubber tubing with an air-pump, such as is used for producing pneumatic massage. The movements are transferred to the probe by the thin diaphragm. It should be placed very lightly against the short process of the hammer, and then very rapid beats can be given.

The Use of Pilocarpine.—Pilocarpine has apparently a marked action in a limited number of cases of acute and recent labyrinthine deafness, but it probably has no effect on chronic or old-standing cases. It is usually given in hypodermic doses of from $\frac{1}{20}$ to $\frac{1}{2}$ of a grain. The patient must remain at home, preferably in one room, during the treatment, and the injections may be repeated every day for a fortnight. Pilocarpine may also be administered in the form of a pill in doses up to $\frac{1}{2}$ gr., and I have recently observed marked improvement after its use in this way in 3 cases of acute labyrinthine deafness. The drug is contraindicated in heart disease, in diseases of the bronchi and lungs, and in the very old and weak. During its administration the patient should be kept under observation.

Bier's Congestion in the Treatment of Ear Disease.—Since the publication of Bier's remarkably successful results in the treatment of acute suppuration by passive congestion, a number of other observers have tried this method. The congestion is produced by applying an elastic bandage round the neck sufficiently tightly to give the face a bluish-red colour and a rather turgid appearance; the bandage remains on twenty-two hours out of the twenty-four. The skin of the neck should be carefully attended to in order to avoid ill effects from the pressure of the bandage. The general opinion is that the method has not much scope in severe forms of ear disease. Experience has shewn that it is contra-indicated in arteriosclerosis, in the presence of intracranial complications, that it is probably dangerous in pneumococcal cases, and that it may be impracticable in children with adenoid vegetations. From a critical study of this method of treatment, Fröse considers that it is of value in slight cases of acute uncomplicated middle-ear suppuration, and also in cases of subacute and not too recent inflammation of the mastoid process in which a subperiosteal abscess has formed before the congestion is induced. He has also obtained benefit in cases of chronic suppuration. Further observation is required before it can be finally decided in what forms, if any, of otorrhoea this method is specially indicated. When congestion treatment is applied the pain certainly seems to disappear rapidly; but in some cases it is the symptom only which is influenced, the inflammatory process continuing unchecked. Heymann discusses whether it is possible to induce a real congestion of the middle-ear cavities in this way, and from his observations it would appear to be improbable. Finally, it should be remembered that during the treatment the patient must not be allowed to go about, and should usually be kept in bed, whilst the utmost watchfulness on the part of the surgeon is essential.

Suction in Aural Disease.—Reference must be made here to the method of applying suction in cases of suppurative inflammation in the middle ear with perforation of the drum membrane. Sondermann is the chief supporter though not the originator of this means of treatment. He has devised a mask which covers the auricle and which is connected by tubing to an india-rubber bag, the latter being furnished with a valve. The mask is held firmly against the side of the head; accurate apposition is secured, as the rim is fitted with an india-rubber air-cushion. When the mask is in position the bag is alternately compressed and relaxed; on compression the air escapes through the valve. The treatment can be relegated to the patient, and should be repeated several times a day. It may be used in conjunction with Bier's treatment by congestion.

The Treatment of Tinnitus Aurium.—Tinnitus aurium, one of the commonest and frequently the only symptom of disease of the ear, is unfortunately also one of the most difficult to treat, yet it may in some cases be so severe as to make the sufferer's life almost unbearable. As it occurs in a great number of conditions it is convenient that the treatment should be discussed here.

To cure the disease of the ear which causes the symptom would of course be the correct treatment if that were possible, but something must also be done in the numberless cases in which this is not feasible. It should not be forgotten that psychical treatment is of great importance. Patients should be assured that the condition from which they are suffering is not the result of grave organic disease endangering either life or reason, and that they must make up their minds to put the matter as far as possible out of their thoughts. It will then be found that many will be able to tolerate the symptom without serious discomfort. While psychical treatment must play an important part in the management of such a case, certain local measures should also be taken. Electricity, in the form of the interrupted or the continuous current, may be employed, as described on p. 382. Pneumomassage also gives some temporary benefit in certain cases. Türck pointed out that massage over the mastoid process and also over the prominent seventh cervical spine may be of value. Lucae suggested that a sounding tuning-fork, with a pitch at the opposite end of the scale to that of the tinnitus, should be held in front of the affected ear. Lumbar puncture is discussed below. Lermoyez records the case of a man in whom tinnitus was relieved and finally disappeared as the result of adopting a salt-free diet. Of drugs, bromides are most efficacious, and may be given during acute exacerbations, whilst dilute hydrobromic acid may be administered for longer periods. As a local application chloral hydrate and cocaine have been injected into the middle ear through the Eustachian catheter.

Vertigo, like tinnitus, is met with in a large number of aural diseases, and its severity also varies greatly. The treatment, the results of which are often unsatisfactory, naturally depends on the amount of discomfort experienced by the patient; occasionally even operative interference may be called for. It is always most essential that the patient should lead a strictly regulated life, and be temperate in all things, as indiscretions in diet, undue exertion, worry, may all set up an attack. Of course the local treatment of the ear suitable for each particular case must not be neglected. As regards drugs, bromides may be useful. J. Babinski has tried the effect of lumbar puncture in labyrinthine vertigo and tinnitus; as he considers the procedure to be free from risk, and as the symptom has never been aggravated by the treatment, he thinks the method justifiable. He removed small quantities only of cerebrospinal fluid, 4 to 5 c.c. at a first sitting, then 15 to 20 c.c.; usually, however, only one puncture was made. In 21 out of 32 patients affected with giddiness in which this was tried there was improvement, and in some it was very marked. Out of 90 patients suffering from tinnitus, the symptom diminished or disappeared in 30, the beneficial effect being not nearly so noticeable as in vertigo.

In some patients vertigo is so disabling, and their misery so distressing, that more heroic treatment is called for. This consists in opening and so destroying the function of the semicircular canals and vestibule. Before so drastic a step can be advocated there must be sufficient evidence that the vertigo is really due to local and not to central disease; further, the patient must be prepared to become totally deaf in the affected ear, and he must be warned as to the possibility of failure. An alternative operation is the division of the auditory nerve as it enters the internal auditory meatus. The results of the latter operation have not been so satisfactory, and the risk to life is greater.

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DISEASES OF THE EXTERNAL EAR

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As can be readily understood, many affections of the external ear, such as skin eruptions or new growths, in no way differ from those elsewhere in the body. It will, therefore, be necessary to consider those diseases only which, either in their clinical course or treatment, present special features peculiar to this region. Some affections, such as othaematoma and perichondritis, may be limited to the auricle; others again, for instance otomycosis, furunculosis, and exostosis, to the auditory canal; or, as in the case of eczema, the whole tract may be implicated. In order to avoid repetition, an account will first be given of the conditions which affect the whole of the external ear, and afterwards of those which independently affect the auricle or the external meatus.

Malformations of the External Ear.—The position and size of the auricle vary so considerably that it is sometimes difficult to decide whether the condition presented is merely a variation from the normal or a true malformation. A well-proportioned auricle with a well-marked and uniform helix suggests a high type of development, but in the lower types, as in the criminal classes and in the insane, the auricle frequently shews certain retrograde or atavistic characters. Apart from these minor variations, malformation of the auricle may be either congenital or acquired. In the congenital form the auricle may be completely absent, or be represented only by a rudimentary fold of skin or by cartilaginous tubercles; or, on the other hand, it may more nearly approach the normal. Associated with this deformity there may be auricular appendages, or congenital fistulas, which are usually situated just above and in front of the tragus or along the ascending part of the helix. In addition, there may be complete atresia of the external auditory meatus, with malformation of the tympanic cavity or of the Eustachian tube, asymmetry of the face, or facial paralysis; these pathological conditions are not surprising, seeing that the structures concerned are all developed from the cleft between the first and second branchial arches and their surrounding tissues.

Acquired malformations are usually the result of injury, frost-bite, othaematoma, and perichondritis, or of some specific disease, such as lupus or syphilis.

The *treatment* varies according to the origin and extent of the malformation. A projecting auricle may be controlled by a special head-cap; in slight deformities a plastic operation may be indicated, or in the more severe forms an artificial ear may be adapted. In congenital bony atresia of the external meatus it is unjustifiable, no matter how tempting it may appear, to perform an operation in the hope of making an artificial canal in order to restore the hearing power. Apart from the difficulty of retaining the patency of any canal so made, there is always an accompanying malformation of the middle ear which renders a successful result impossible. Although in a few cases it has been stated that the tympanic membrane was exposed by operation, experience shews that the supposed tympanic membrane was probably the capsule of the temporo-maxillary joint. When both ears are affected, the education of the child is, of necessity, the same as that of a deaf-mute. In these cases, however, the labyrinth is rarely affected, and in consequence the deafness, though marked, is not absolute, so that the child, if carefully trained, may to a large extent overcome the physical disadvantages present at birth.

Eczema of the External Ear.—Eczema of the auricle is usually due to some external irritant. In infants, from tenderness of the skin, it may result from such slight causes as carelessness in drying the ear after washing, or from the use of an irritating soap. In children it is frequently secondary to middle-ear suppuration, especially when tuberculous in origin, and is very common among the poor as the result of uncleanliness, rickets, and unfavourable hygienic surroundings. The younger the patient the greater the liability to the acute forms of eczema, but in adults the dry and chronic forms are more usually met with, especially in the gouty. In acute eczema the auricle becomes red and swollen with the formation of vesicles, which soon burst and leave behind a moist, weeping surface; as this condition has an acute onset, it must be distinguished from erysipelas. In addition there is usually a sensation of heat, tenderness, and intense irritation within the ear. When the external meatus is also implicated, the lining membrane of the auditory canal may become so swollen as to produce complete occlusion and thus give rise to temporary deafness. The secretion, which at first is serous, may, from infection, afterwards become purulent, and be so copious as to simulate suppuration of the middle ear; this, however, can be excluded by careful examination of the tympanic membrane, which will be found to be intact. The hearing power, provided the auditory canal is not completely occluded, remains good.

Treatment depends upon the cause and the condition found. When the cause of the irritation is purely local, simple measures are usually sufficient, though general treatment must not be neglected. In children, for instance, special attention must be paid to diet, to placing them in the best possible hygienic surroundings, and to improving their general health by tonics, such as syrup of the iodide of iron, or liquor arsenicalis. In adults, the possibility that this affection is associated with some constitutional disorder must always be borne in mind; for it is as important to treat the general as the local condition. In the acute stage soothing applications are advisable, and syringing or washing the ear with water must be avoided. Thus, the part may be dusted with a powder containing one part of calomel, two parts of zinc oxide, and three parts of starch; or, if there be much irritation with redness and tenderness of the auricle, compresses of a weak solution of glycerin of subacetate of lead, to which a few drops of tincture of opium may be added, are most serviceable. Crusts should be softened by oil containing a dram of nitrate of mercury ointment to the ounce, and then removed; the auricle being afterwards protected with lint soaked in calamine lotion, or smeared with an ointment of lead and mercury. The chronic conditions demand more stimulating treatment, and much patience and the application of various ointments and lotions may be necessary before cure is finally obtained. Amongst the remedies most advocated are ointments containing resorcin, benzoate of zinc, ichthyol, and, according to Sagabiel, naphthalin. Much benefit is often derived from painting the affected surface with almond oil containing 2 per cent of oil of cade, or with a 2 to 5 per cent aqueous solution of silver nitrate. Although treatment is usually followed by complete recovery, in some of the more chronic cases considerable thickening of the subcutaneous tissues eventually gives rise to narrowing of the auditory canal or deformity of the auricle.

Herpes zoster of the external ear is comparatively rare. The site of the affection depends on whether the auriculo-temporal or the great auricular nerve is affected. In the former case, groups of vesicles are found on the anterior surface of the auricle and anterior margin of the external meatus; in the latter, on the posterior surface. The onset is characterised by sudden and intense neuralgic pains in the ear, which may radiate to the surrounding parts, but usually subside after two or three days, on the appearance of the herpetic vesicles. With this there may be considerable swelling and redness of the auricle at the site of the affection, and as a rare condition vesicles have also been observed on the tympanic membrane itself. Facial paralysis is not infrequently associated with herpes of the ear, generally appearing either with or shortly after the appearance of the vesicles. Sarai records an interesting case in which, three days after the eruption of the vesicles, facial paralysis occurred, together with loss of taste on the anterior third of the tongue on the affected side; and Körner another in which both the facial and auditory nerves were affected.

The principles of *treatment* are to alleviate the pain by anodynes, such as an oil containing menthol, carbolic acid, and cocaine; or, if this prove ineffectual, by a local subcutaneous injection of morphine. In addition, the ear should be protected by means of a soft pad of lint or wool. After the vesicles have ruptured, the affected part may be dusted with some inert powder, such as kaolin. Of internal remedies the most serviceable are antipyrin, aspirin, or pyramidon. As a rule, the vesicles and other inflammatory symptoms disappear by the end of the second week, but neuralgic pains may continue for a considerable period, just as intercostal neuralgia may persist after herpes of the chest wall. On the other hand, facial paralysis, when it occurs, may not shew any sign of improvement even for months, but usually there is complete recovery.

Syphilis of the external ear has been known to occur as a primary lesion of the auricle, the result possibly of a kiss or a bite, but it is more frequently observed as a secondary or tertiary lesion. In secondary syphilis the auricle and auditory canal may be attacked, but comparatively rarely, and then only as a part of the skin eruption simultaneously affecting other parts of the body. Condylomas, from diffuse swelling of the walls, may partly or completely occlude the auditory canal, and may exude a slight secretion which, if ulceration occur, may become more profuse and fetid. There may also be considerable pain within the ear, and tenderness on movement of the auricle, or during mastication, so that, but for other signs of syphilitic infection, the condition may easily be mistaken for a simple diffuse inflammation of the external ear. In addition to ordinary antisyphilitic treatment, the auditory canal should be syringed with a weak solution of perchloride of mercury, the meatus being afterwards dried and lightly plugged with gauze impregnated with calomel. As a rule, complete recovery takes place, but in some cases, in consequence of the deep ulceration, or perhaps from unwise cauterisation, cicatrisation and subsequent stenosis of the external meatus may occur. In the tertiary lesions, gummas of the auricle may cause deep-seated destruction, with a resulting perichondritis and considerable deformity, which in certain cases may closely simulate lupus or squamous-celled carcinoma.

Lupus as a primary affection of the external ear is exceedingly rare. It is, however, more common in association with lupus elsewhere in the body, and most frequently occurs as a direct extension of lupus of the face. It may be met with in all its various forms, which are, as a rule, similar in character to those simultaneously affecting other parts. Sometimes, except for the presence of groups of tubercles and nodules, it closely resembles chronic eczema of the auricle; in other cases the ulceration is so extensive and so deep that the diagnosis from malignant disease is exceedingly difficult. Again, when the lobule alone is affected, it not infrequently appears as a large pendulous mass resembling a fibroma. In the severer forms the destruction and deformity of the auricle may be very great, largely because lupus is apt to attack the cartilage. Sometimes the upper part of the auricle is completely destroyed, and if seen after scarring has taken place, may resemble the results of frost-bite. Occasionally cicatricial tissue may bind down the remnant of the auricle to the side of the head, and completely obliterate the entrance of the external auditory meatus.

To obtain a cure is difficult, and in some cases impossible, although light treatment, according to the principles laid down by Finsen, has given surprisingly good results. If the disease is not too extensive, light treatment should undoubtedly be recommended. In many cases it apparently cures, although experience has shewn that all lupus cases are apt to relapse. In more extensive disease the treatment may be considerably curtailed and rendered more effective by first curetting away exuberant granulations under a local or general anaesthetic. More recently, inoculation treatment, as advised by Sir A. E. Wright, has proved most successful in the more acute conditions, especially when the opsonic index is extremely low. When the diagnosis is doubtful and malignant disease is suspected, seeing that it is possible for an epithelioma to be grafted on the lupoid condition, a small portion of the diseased area should be excised for microscopic examination.

Malignant disease of the external ear is also comparatively rare. It is usually a squamous-celled carcinoma or a rodent ulcer; sarcoma and endothelioma are excessively rare, and as a rule are confined to the auditory canal. In its early stages squamous-celled carcinoma of the auricle seldom gives rise to any special symptoms, and for this reason is often in a somewhat advanced condition before advice is sought. It usually occurs in elderly persons, and not uncommonly originates in an apparently benign wart or fissure which may have existed for years. Its most frequent site is the upper portion of the pinna. The chief symptom from which the patient suffers, apart from the fetor and the inconvenience of the ulceration, is intense radiating pain, which may at times be agonising, especially when the deeper parts become invaded. The diagnosis is based on the clinical appearance and on microscopic examination. In the early stage, before infection of the pre-auricular and cervical lymphatic glands, it is possible to obtain a cure by complete excision of the affected part; in the later stages the growth may extend into the external meatus, or to the surrounding parts, and with this, sooner or later, the cervical glands become infected. In such cases, although it is too late to hope for eradication of the disease, operative measures may still be justifiable if there is much pain, as there is no doubt that in many of these cases considerable relief is thus obtained. Squamous-celled carcinoma of the external auditory canal, although almost invariably occurring as an extension of the growth from the auricle or tympanic cavity, may also be primary.

Rodent ulcer is more amenable to treatment than the other forms of malignant disease, on account of its slow growth and the absence of infection of the cervical glands; successful results have been repeatedly obtained by *x*-ray treatment.

In an elderly patient the presence in the auricle or auditory canal of an ulcer or granulations which resist treatment, or recur repeatedly after removal, suggests malignancy; and as the only chance of eradicating the disease is complete removal of the affected part by operation before the deeper structures and lymphatic glands become invaded, a fragment of the suspected growth should be submitted to microscopic examination without delay.

Neuroses and reflex nervous phenomena in connexion with the external ear, being occasionally met with, may be briefly mentioned. An accumulation of cerumen, the presence of a foreign body within the auditory canal, the mere insertion of an aural speculum or the syringing of the ear, even though the tympanic membrane is intact, have all been known to give rise to cough, tinnitus, attacks of vertigo, or to epileptic seizures, these reflex phenomena being probably due to irritation of the auricular branch of the pneumogastric nerve. Thus, Lannois records a case of a girl who was completely deaf on both sides and had signs of pseudo-meningitis and paraplegia of the extremities for two years; all the symptoms rapidly disappeared after removal of a large mass of cerumen in which a needle was embedded. In one case hoarseness and deafness were cured by removal of a piece of a carpenter's pencil from the ear; in another case, in which attacks of tinnitus and severe vertigo were due to a fungus, *Aspergillus niger*, within the ear, recovery took place after the condition had been cured.

Otalgia of the auricle may also occur, and is usually confined to the anterior or posterior surface, corresponding to the distribution of the auriculo-temporal or of the great auricular and small occipital nerves.

Again, intense pruritus of the auditory canal, without any objective symptoms, and so presumably of nervous origin, is met with in rare instances, although the majority of such cases are associated with diminished secretion of cerumen.

Anaesthesia is seldom confined to the auricle, being usually part of a general hemi-anaesthesia of the head. In other cases the affection is apparently of vasomotor origin. These phenomena, as might be expected, are generally associated with hysteria or its allied conditions. Careful examination should, nevertheless, always be made to exclude any organic lesion, more especially cerebral disease.

No definite line of treatment can be laid down, as this largely depends on the origin of the affection. Locally, anodynes or blisters may be applied over the painful areas. In other cases internal administration of sedatives, tonics combined with massage, a rest cure, or galvanism are of benefit; but, as a rule, unless an organic lesion be discovered the case is a source of great anxiety to the patients and their friends, and one of extreme worry to the practitioner.

DISEASES OF THE AURICLE.—**Haematoma of the auricle (Othaematoma)** is an effusion of blood either into the substance of the cartilage or between it and the perichondrium. Although usually the result of injury, such as laceration of the cartilage or rupture of the subperichondrial blood-vessels in consequence of direct violence, it occasionally appears to be spontaneous, but even then it is often doubtful whether injury has not really had some share in its production. It is quite conceivable that as a result of degenerative changes in the cartilage or blood-vessels, a very slight injury might be sufficient to produce a haematoma. Meyer states that as age advances the cartilage softens and becomes fissured, and that its substance may shew small areas containing homogeneous gelatinous material and very vascular connective-tissue. This statement bears on the relative frequency of haematoma of the auricle among the insane, although here again injury is probably the exciting cause and the pathological conditions merely disposing. Against this conception of degeneration it may be urged that spontaneous haematoma is not necessarily confined to adults; for instance, Mignon records such a case in a child aged nine, in which the differential diagnosis from a cyst had to be made by puncture.

Symptoms.—When it occurs spontaneously there may be no marked symptoms; but when it follows a blow, as may occur in football-players or pugilists, pain and other signs of inflammation are usually present. In a typical case the auricle is swollen by a semi-fluctuating, irregular tumour, filling up the surface of the concha, and often of a purplish or bluish tint and dark to transmitted light.

The treatment of othaematoma in the earlier stages is chiefly expectant, and may be limited to compresses of evaporating lead lotion and the application of gentle pressure to the auricle by means of a bandage. If strict asepsis can be guaranteed, Passow advises early incision and evacuation of the blood-clot. If suppuration occur, this becomes essential to allow of escape of the purulent contents; the condition should be treated as an ordinary abscess, curetting away, if necessary, any granulation tissue or necrosed parts of the cartilage. The wound should be thoroughly cleansed with a solution of hydrogen peroxide, and afterwards gently irrigated with biniodide of mercury or carbolic lotion. The resulting deformity is often considerable, and is determined by the amount of destruction of the cartilage. In some cases, however, after the acute symptoms have disappeared, massage of the auricle may be advised in the hope of reducing the deformity caused by thickening of the auricle.

Perichondritis or inflammation of the cartilage of the auricle, like othaematoma, may occur spontaneously without any obvious cause, although it is most frequently due to infection as a sequel of furunculosis of the external meatus; or of some operation on the cartilage of the ear, as in the formation of the post-meatal skin-flaps in the complete mastoid operation. It is also occasionally syphilitic or tuberculous, or is the result of some acute circulatory disturbance, such as frost-bite. More recently Lermoyez, Leutert, and others have drawn attention to the presence of the *Bacillus pyocyaneus*, which has been repeatedly found in pure culture, and their observations point to the possibility that this bacillus may attack the cartilage in the absence of any obvious abrasion of the skin.

The symptoms vary with the cause; in the idiopathic and syphilitic forms the onset is much less acute than in those cases in which it is the result of pyogenetic infection. The inflammation, which in the earlier stages may be mistaken for furunculosis, usually begins at the entrance of the auditory canal. The inflammatory swelling gradually spreads over the inner surface of the concha, eventually implicating the whole auricle, which from swelling of the surrounding soft tissues may project somewhat from the head. There may also be considerable pain and fever. The inflammatory exudation between the cartilage and the perichondrium is at first of a serous or synovial character, so that the auricle is translucent to transmitted light, thus distinguishing it from othaematoma. In benign cases the inflammation and fever may subside after a few days; but in the more serious cases, after ten to fifteen days, a fluctuating area may be felt in the region of the concha as the result of suppuration. When due to infection by the *Bacillus pyocyaneus*, the pain is often very severe, and if there be any discharge from the ear it is frequently of a bluish-green colour, which, however, gradually disappears as recovery takes place.

The treatment is practically that of haematoma. In the spontaneous form it is usually sufficient to protect the ear and to apply soothing lotions; unless absolutely necessary incisions into the auricle should be avoided, as the inflammation not infrequently subsides without any suppuration. If due to syphilis or tuberculosis, these conditions must, of course, be treated. When the result of pyogenetic infection every care should be taken to render the parts as aseptic as possible, the line of treatment being that already laid down for suppurating othaematoma. As the *Bacillus pyocyaneus* is essentially aerobic, solutions of hydrogen peroxide should be avoided when it is present; in such cases the wound should be packed with gauze soaked in a solution of a two to five per cent of silver nitrate. As in othaematoma, the resulting deformity is often considerable; although in benign cases, if no suppuration takes place, the auricle may gradually resume its normal appearance.

Gangrene of the auricle may occur either as a purely local affection and as a superficial gangrene of the pinna, or as a very serious affection, noma, which is always accompanied by grave constitutional symptoms.

Superficial gangrene is usually the result of frost-bite, but it sometimes occurs symmetrically in Raynaud's disease. In frost-bite the ear should be protected by pads of aseptic wool, and after the line of demarcation appears the gangrenous portion may be excised. In Raynaud's disease the general condition should also be treated.

Noma of the auricle is fortunately exceedingly rare, and is usually only met with in marasmic infants in unhealthy surroundings, and suffering from middle-ear suppuration, which in these cases is not uncommonly a sequel of measles. The gangrenous condition usually begins within the auditory canal, from which exudes a foul discharge. It rapidly spreads to the auricle, and perhaps also to the surrounding parts, which become swollen, of a darkish hue, and eventually black. It is always accompanied by the most grave constitutional symptoms of toxaemia, and unless the most energetic measures are adopted almost invariably terminates fatally. The child must be kept warm, and nourishment, restoratives, and stimulants frequently given. Locally, the best possible chance of recovery lies in excision of the affected part with free use of antiseptics.

Benign Growths.—In addition to malignant disease already mentioned, the auricle may be the seat of benign growths of every variety. As a manifestation of gout, *tophi* along the margins of the helix, concha, or other parts of the auricle are not uncommon. In this connexion Prof. Osler records three cases of *calcification* of the cartilage, presumably due to deposition of biurate of sodium which, however, did not invade the overlying skin. *Ossification* of the auricle has also been recorded in a lunatic whose ear had been frequently injured. *Fibromas*, usually found in the lobule, are due to irritation from the insertion of an ear-ring; more rarely fibrochondroma is seen. *Horny excrescences*, sometimes of considerable size, also occur, and are probably due to the overgrowth of an everted sebaceous follicle. True *tuberculosis* of the auricle is rare, and usually appears as cutaneous ulcers or perichondritis, but it may also occur as a nodular mass on the lobule, closely simulating a simple fibroma. Cysts, angioma, angiolipoma, and cirsoid aneurysm are rare.

DISEASES OF THE EXTERNAL AUDITORY CANAL.—The affections of the external meatus may be grouped into those causing obstruction of its canal, and those giving rise to inflammation of its walls. Amongst the former may be mentioned foreign bodies, impacted cerumen, stenosis and atresia, and exostoses; amongst the latter, furunculosis, general diffuse inflammation of the auditory canal, otomycosis, and diphtheria.

Foreign bodies are most frequent in children, who may deliberately push them into the ear; in adults their occurrence is less frequent, and usually accidental, except when they have been inserted for a specific purpose, such as the centre of an onion to relieve earache, or a pledget of wool or an artificial ear-drum, which may have been introduced and forgotten. It is extraordinary what various kinds of foreign bodies may be met with: for example, living creatures, such as flies, earwigs, maggots, or cockroaches; vegetable substances, as a pea, bean, or an ear of corn; or a hard substance, as a button, fruit-stone, or piece of slate pencil. An inert foreign body may remain for an indefinite period, even for twenty-five years, without giving rise to any symptoms, except, perhaps, to deafness, which is especially apt to occur from an accumulation of wax around the foreign substance. On the other hand, the reflex irritation may be sufficient to give rise to attacks of coughing, vomiting, tinnitus, and vertigo. Living insects, by their scratching movements against the drum, may set up the most intense pain and tinnitus. From injury to the walls of the external meatus, especially if the foreign body is hard and of irregular outline, acute inflammation of the external meatus may occur, and is usually accompanied by a purulent discharge, which does not cease until the foreign body has been removed. Serious complications are rare, and almost invariably the result of injudicious attempts to remove the foreign body by means of instruments, unfortunately often without the assistance of artificial light and a speculum. In consequence of such manipulations the tympanic membrane may be injured, and cases are recorded in which there has been subsequent middle-ear suppuration, acute inflammation of the mastoid process, or even meningitis from direct injury of the inner wall of the tympanic cavity.

It is first necessary to make a careful inspection of the ear in order to see that a foreign body really is present, as the statements of a patient, especially if of a nervous temperament, are not always trustworthy. It is also important to determine its size, character, and position before deciding what treatment to adopt. In most cases the foreign body can easily be expelled by simple syringing of the ear, the stream of lotion being directed towards any chink which exists between it and the wall of the meatus. Living insects can at once be killed by the instillation of a few drops of oil, or rectified spirit, and then easily removed; if very large, like a cockroach, it may be so firmly impacted within the meatus that it can only be extracted by means of forceps. Sometimes the insect is so tiny that it lies hidden in the sinus of the external meatus and escapes detection even after a thorough inspection of the auditory canal, but its presence may be suspected from the agonising tinnitus and pain produced by its movements on the drum.

If there is much inflammation and swelling of the walls of the external meatus, often due to previous ill-advised attempts to extract the foreign body, it is advisable to wait until the inflammation has subsided before attempting its removal, and in the meanwhile to prescribe frequent instillations of an alcoholic solution of biniodide cf mercury. When, however, the foreign body is a vegetable substance, such as a pea, the moisture in the ear is apt to make it swell, and for this reason the longer it is left in the ear the more difficult will its removal become. If, in these cases, syringing prove ineffectual, ear-baths of rectified spirit may be given, as they will tend to cause shrinkage of the vegetable substance. After two or three days, further syringing will probably prove successful. If repeated attempts at syringing fail to remove the foreign body, its extraction by means of an instrument may be indicated, especially if it has been pushed in beyond the isthmus or narrow region of the auditory canal at the junction of its cartilaginous and bony portions. Another condition requiring its immediate removal is acute inflammation of the middle ear, accompanied by signs of retention of pus. The method of removal depends on the age of the patient and the character of the foreign body. In an adult no anaesthetic may be necessary, but the head should be firmly supported to prevent movement during the attempts at extraction. In children it is advisable to give a general anaesthetic. The instruments used consist of tiny hooks, and scoops and forceps of varying sizes and shapes. A clear view of the auditory canal must always be obtained by means of reflected light and the insertion of an aural speculum. In these manipulations special care must be taken not to push the foreign body farther in, and not to injure the walls of the auditory meatus or tympanic membrane. After extraction the meatus should be carefully inspected. If the tympanic membrane and auditory canal have not been injured, it is sufficient to dry the meatus and puff in a little boracic powder. If, however, the tympanic membrane has been wounded, middle-ear suppuration may result, which must be treated by the ordinary methods. Occasionally these attempts to remove the foreign body are unsuccessful, and, in order to obtain more room, it may be necessary to reflect forward the auricle by a post-aural incision so as to expose the bony meatus freely. This operation is especially indicated when the foreign body has been pushed into the tympanic cavity, or when its removal becomes imperative on account of signs of retention of pus within the middle ear. If, in these cases, there is, in addition, chronic middle-ear suppuration, the complete mastoid operation may be advisable. It is, however, impossible to lay down definite rules for these various surgical procedures, as each individual case must be treated on its own merits.

Impacted Cerumen.—The cerumen, or wax, which is constantly secreted in small quantities by the ceruminous glands, in normal circumstances is gradually removed as it is formed by movements of the jaw or in the process of cleansing the ears. Any condition which hinders this, such as a stricture of the auditory canal or an abnormally narrow entrance of the meatus, may be considered a disposing cause to its accumulation. It may also be due to excessive secretion, or to hyperaemia of the walls of the meatus, as in chronic eczema, when the plug is frequently largely composed of epithelial debris; or the whole mass may consist of desquamated epithelium merely coloured by cerumen, and containing cholesterin crystals, a condition sometimes spoken of as keratosis obturans.

Impacted cerumen is, in itself, such a trivial matter, and so common, that it seems almost unnecessary to mention it. At the same time, a simple accumulation of wax may cause the most unpleasant symptoms, and even mistakes in diagnosis. Thus, the irritation due to the pressure of impacted cerumen may be sufficient to cause the most distressing tinnitus, or even attacks of vertigo and epilepsy. Cases, indeed, are not unknown in which a plug of wax has been overlooked, and the deafness has been diagnosed as due to some incurable form of middle-ear catarrh.

The symptoms vary very much, and, beyond occasional tickling or discomfort in the ear, nothing may be noticed until the cerumen completely obstructs the auditory canal. This may occur very gradually, so that the resulting deafness may be only discovered accidentally. As a rule, the final and complete obstruction of the auditory canal occurs suddenly, often in consequence of water getting into the ear and causing rapid swelling of the ceruminous mass. The symptoms are then more pronounced, as, in addition to deafness, there is usually a feeling of fulness in the head, reverberation of the voice on the affected side, tinnitus, and giddiness. Occasionally there is also earache, but this is generally due to concomitant inflammation of the middle or external ear.

Prognosis.—It is not wise, on the discovery of cerumen, to assure the patient that recovery of hearing will at once take place on its removal; sometimes no benefit arises, because there is, in addition, disease of the middle or internal ear, and it has, indeed, happened that the removal of cerumen has further impaired the hearing, owing to the pre-existing plug having covered a perforation of the tympanic membrane and acted as an aid to sound conduction. Also, the patient should be warned that an accumulation of wax is apt to recur, especially if the auditory canal be narrow or affected by eczema.

Treatment.—In the majority of cases simply syringing the ear is sufficient to remove the plug of wax. It is advisable, however, first to make certain by careful inspection that cerumen really is present. If this is not done as a matter of routine, mistakes are apt to be made. The unfortunate practice of syringing the ear because the patient happens to be deaf has, in many cases, led to disaster. The symptoms may possibly be due to middle-ear catarrh, or to a previous middle-ear suppuration with an existing perforation of the tympanic membrane, in which case syringing will be quite useless and perhaps harmful; on the other hand, it is not always easy to remove a plug of wax at the first attempt, so that it does not necessarily follow that the inability to extract a plug of cerumen means that the auditory canal is quite clean. Careful inspection, however, of the ear, both before and after syringing, will prevent such errors. If a plug of wax can be easily removed by syringing, it is sufficient, after its removal, to dry the ear and protect it, for a day, with a small piece of wool. If, however, the wax cannot be so removed, it is wiser first to soften the mass by the instillation of some drops containing a dram of glycerin and 30 grains of bicarbonate of sodium to an ounce of water. This should be done at bed-time, and the next morning the ear should again be syringed. Occasionally, after removal of the cerumen, the tinnitus and feeling of fulness in the head do not disappear; this is usually due to retraction of the drum from the pressure of the cerumen. Provided earache and the other signs of inflammation of the middle ear are absent, gentle inflation of the ears is not contra-indicated, and will probably completely relieve these symptoms.

Stenosis of the external meatus, whether occurring as complete atresia or as partial stricture of the meatus, is only of medical interest in that it may give rise to impairment of hearing or dispose to those dangerous sequels which may occur in the course of a middle-ear suppuration. As has already been mentioned, congenital atresia of the external meatus is usually associated with deformity of the auricle, and complete obliteration of the auditory canal with malformation of the tympanic cavity. The chief causes of acquired stenosis or atresia are injury, inflammation, and ulceration or necrosis of the walls of the auditory canal, the latter being frequently the result of diphtheria or scarlet fever in childhood. Partial stenosis is usually only discovered accidentally, advice being sought on account of eczema or some other aural affection.

Treatment.—In congenital atresia an operation is unjustifiable (*vide* p. 390). If acquired, operative measures may be advised, provided the atresia be limited to the outer part of the canal and it seem probable that the middle ear is intact. In stricture of the meatus no treatment is necessary unless middle-ear suppuration coexists. In acute suppuration cure may follow simple conservative measures. If the condition is chronic, and from time to time there are symptoms of retention of the purulent discharge, the only treatment to be recommended is the complete mastoid operation, special care being taken so to fashion the meatal skin-flaps as to prevent recurrence of the stenosis.

Exostosis and hyperostosis of the external meatus, like partial stricture of the auditory canal, give rise to no symptoms until, from complete obliteration of the auditory canal, they cause deafness; or they may be discovered accidentally during the examination of the ear for some other aural affection. Nothing certain is known as to their origin. When they are bilateral, and especially if they occur close to the tympanic membrane at the anterior and posterior margins of Shrapnell's membrane, that is at the margins of the tympanic ring, they may possibly be due to some abnormality of development. The frequency of hyperostoses or diffuse thickenings of the meatal canal in gout or rheumatic subjects suggests some causal relation, and they are by no means uncommon in the condition known as otosclerosis. Not infrequently they are also met with as a result of syphilis, both in the acquired and in the inherited forms. Further, any condition, such as chronic suppuration of the middle ear or chronic inflammation of the external ear, which may lead to inflammation of the periosteal lining of the auditory canal, will favour their production. They vary in their consistence, situation, and number; and their growth, as a rule, is exceedingly slow. As their surfaces come in contact, in addition to causing deafness from complete occlusion of the meatus, they may, from pressure against one another, give rise to an acute inflammation of the skin covering them, or even to ulceration with purulent discharge; a condition which may closely simulate furunculosis. On examination, the lumen of the meatus may be found to be almost completely obstructed by a single pedunculated growth, yellowish-white in appearance, hard, and almost insensitive when touched with a probe; or several small growths may stand out prominently as slight projections from the meatal wall; or the meatus may merely have the appearance of narrowing, the growth having no definite outline and fading away into the surrounding tissue.

Treatment.—If the narrowing of the meatus is not very great and the growth has only been discovered accidentally, there is no immediate necessity to take any active measures. The patient, however, should be warned of his condition, and should be informed of the importance of at once obtaining medical advice if, at any future period, he becomes deaf or has earache; symptoms which may be the result of an accumulation of wax or perhaps may be due to inflammation of the external or middle ear. Concomitant gouty, rheumatic, or syphilitic conditions should, of course, be treated.

If there is pain from pressure of the exostoses against one another, immediate treatment should be directed towards its alleviation, such as the frequent instillation of a few drops of a 10 per cent solution of carbolic acid and glycerin. After the acute inflammation has subsided, drops containing ten grains of boric acid or menthol to an ounce of rectified spirit may be prescribed in the hope of causing further diminution of the swelling of the soft tissues. Occasionally, as the result of this treatment, a sufficient opening can again be obtained within the auditory canal, so that the hearing once more becomes normal. Relapses, however, are prone to occur with renewal of the pain and other symptoms of inflammation.

In all cases of exostoses and hyperostoses, the question of operative treatment will have to be considered. It is justifiable and indicated in the presence of middle-ear suppuration on the affected side and signs of retention of pus from narrowing of the meatus, or when the pressure of the exostoses produces pain which cannot be relieved. If the exostoses nearly block up the external meatus of both ears, and there is every prospect of each side becoming completely obstructed in the near future, an operation may be advised on the worse side. When one ear only is affected, and there are no other symptoms except deafness and obstruction of the external meatus on the affected side, the question of operation may be discussed with the patient, if a person of intelligence, who can then decide for himself whether he will wait until other symptoms arise or undergo immediate operation. Before operation is advised a careful examination should be made in order to exclude the possibility that the deafness is due to chronic middle-ear catarrh or disease of the internal ear, as in these conditions the result of the operation would be negative. Formerly the exostoses were removed by means of a dental burr, which was inserted into the auditory canal in order to force a passage through the obstructing growth. This method, however, has now been discarded, as it frequently led to disaster, partly from lack of control over the instrument and partly because subsequent stenosis of the canal resulted from cicatrisation of the soft tissues. Two methods of operation may be recommended. If the exostosis is pedunculated and situated in the outer part of the auditory canal, it may be conveniently removed through the external meatus by chiselling through its pedicle. If, however, the exostoses are multiple or have a broad base and are deeply situated, this method is impossible. In such cases the auricle must be reflected forward, in the same manner as for removal of a foreign body, in order to expose the bony meatus freely. If the growth is situated on the posterior wall, the membranous portion of the auditory canal should be separated from it as far as is possible, the exostoses being then removed by means of the chisel. If they are more deeply situated it may be necessary to chisel away part of the posterior wall of the external meatus before attempting to remove them, care being taken not to expose the antrum nor to injure the tympanic membrane. When the growth arises from the anterior wall its removal is more difficult, as the membranous portion of the posterior wall must be freely incised before exposing the anterior wall. This operation should not be lightly undertaken, as the results are not always satisfactory. In the case of a pedunculated exostosis, a certain cure can usually be guaranteed. In multiple growths, especially if deeply situated, there is, in spite of the greatest care, considerable risk, not only of injuring the tympanic membrane, but also of subsequent stenosis of the auditory canal.

Inflammation of the External Meatus.—On account of the close connexion of the skin with the underlying tissues of the membranous and bony meatus, acute inflammation of the auditory canal is one of the most painful affections which the aurist has to treat. It may occur in the circumscribed form as furunculosis or boils, or, on the other hand, as a general diffuse inflammation of the lining membrane.

Furunculosis is due to the infection of one or more hair-follicles by staphylococci, which are usually introduced either through an abrasion of the skin, such as by scratching of the ear with a hair-pin, or as a sequel of middle-ear suppuration. It is not infrequently observed as part of a general furunculosis, in diabetics, or as the result of excessive use of drugs, such as bromides. The onset is sudden; at first there is a feeling of discomfort and fulness within the ear; this rapidly changes to that of acute pain, which may become agonising and throbbing, frequently radiates over the side of the head, and is sometimes so intense as to render the patient almost distracted. It is usually worse at night, and the tenderness of the auricle may make it impossible for the patient to lie on the affected side. The physical signs vary exceedingly; in some cases there are no external signs, except some redness or swelling at the entrance of the meatus; in other cases the auricle is also red and swollen and projects from the head, with considerable oedema of the surrounding parts, implicating even the side of the face and eyelids; or there may be pitting of the skin over the parietal or mastoid region, apparently out of all proportion to the cause; this, if it occur during the course of a middle-ear suppuration, may easily be confused with acute inflammation of the mastoid process. There is also extreme tenderness on movements of the auricle, especially low down in front of the tip of the mastoid process; movements of the jaw may be so painful as to render mastication almost impossible, and not infrequently there is also marked enlargement of the pre-auricular glands. Inspection of the auditory canal may be impossible on account of the extreme pain produced by insertion of a speculum, but if this is successfully accomplished one or more discrete swellings are seen partially or completely obstructing the meatus, and exquisitely tender on probing. Within two or three days after the onset, although sometimes much later, points of pus appear on the summits of the swellings. On bursting of the furuncles there is at once considerable relief of the pain with rapid diminution of the swellings. Unless the meatus is completely obliterated, the hearing power is seldom affected; this point is of practical importance in connexion with the question of diagnosis.

Diffuse inflammation of the external meatus may depend on the same causes as furunculosis, which it may accompany or give rise to; and is by no means an uncommon sequel of eczema of the ear. In furunculosis the cartilaginous portion of the meatus is chiefly affected because the hair-follicles are confined to this region, but in diffuse inflammation the osseous portion and the outer surface of the tympanic membrane are specially prone to become affected. The symptoms are not so acute as in furunculosis, the pain being more deep-seated and aching in character and accompanied by a feeling of irritation and heat within the ear. Frequently, however, there is tenderness on mastication or on movements of the auricle. Examination may shew considerable narrowing or even occlusion of the auditory canal due to a uniform swelling, especially of its deeper parts. In the earlier stages there may only be slight congestion of its walls; but later, desquamation of the epithelial lining of the canal and perhaps also of the outer layer of the tympanic membrane may occur, accompanied by a serous exudation, which eventually may become purulent. Sometimes, in streptococcal infection, the inflammation may be so intense as to invade the periosteum and perichondrium, perhaps ending in perichondritis of the auricle, or in necrosis of small fragments of the bony walls of the auditory canal. In such cases, in addition to the local symptoms, there may be pyrexia, and the patient may feel and look really ill.

Diagnosis.—A furuncle may have to be distinguished from an exostosis, especially if the skin over the latter is inflamed; from a parotid abscess which has opened into the external meatus through one of the fissures of Santorini; or from granulations and polypi within the auditory canal. If the whole of the external meatus and outer surface of the drum become lined with septic granulations accompanied by a purulent discharge, the condition may have to be distinguished from an acute middle-ear suppuration; and if, as in the more severe cases, the auricle also becomes swollen and projects from the head with oedema of the surrounding tissues, the appearance may closely resemble that produced by acute inflammation of the mastoid, or perichondritis of the auricle.

As in other conditions of acute inflammation of the ear, the *treatment* is chiefly expectant, and is directed towards the alleviation of the pain. Warm drops containing two grains each of cocaine, morphine, and carbolic acid in a dram of olive oil may be frequently instilled into the ear. If the symptoms, instead of abating, become more urgent, it may be necessary to incise the furuncles. The pain of such an incision is intense and may produce considerable shock, so that, if possible, this small operation should be performed under a general anaesthetic, preferably gas and oxygen; as a means of deadening the pain, local anaesthesia is quite ineffectual. If the patient refuse to allow incision of the furuncles, leeches may be applied in front of or behind the ear, according as the furuncles are situated on the anterior or posterior wall, and in addition considerable relief of pain may be obtained by frequent syringing with very hot lotions. After the furuncles have burst or have been incised, the meatus may be gently plugged with a small strip of gauze soaked in a 10 per cent solution of carbolic acid in glycerin, or in an alcoholic solution of perchloride of mercury to which a little cocaine may be added. Since other hair-follicles are liable to become infected, it is advisable, as a prophylactic measure, to smear the auditory canal occasionally with an oil containing mercury for a considerable period after cure has apparently been obtained. Granulations within the meatus, due to furunculosis, should be curetted away under a local anaesthetic of cocaine, and if necessary their surfaces should be touched with silver nitrate stick or trichloracetic acid. When there is furunculosis elsewhere every attempt should be made to improve the general condition of the patient, and in the more severe cases injections of an antistaphylococcic vaccin, in accordance with the principles laid down by Sir A. E. Wright, may be given.

In diffuse inflammation of the auditory canal the acute symptoms usually subside under simple aseptic treatment and the use of anodynes. Scarification of the canal is seldom effectual. When the epithelium has desquamated in large patches with the formation of granulations and a purulent discharge, the ear should be cleansed daily by means of an ear-bath of a solution of hydrogen peroxide and afterwards syringed out with a weak antiseptic solution. In the milder cases it is sufficient to puff in a little boracic acid powder and plug the ear gently with gauze. As the condition improves, any remaining raw patches or granulations may be touched with some cauterising agent, such as silver nitrate or fused chromic acid. In other cases a good result is obtained by painting the surface of the meatus with a 10 per cent solution of silver nitrate, by a 1 in 500 or stronger solution of oil of thyme, or by the instillation of rectified spirit containing two or three grains of salicylic acid to the ounce. Unless quickly cured this condition is extremely apt to become chronic, with ultimate thickening of the walls of the meatus and stenosis of the auditory canal; or it may pass into a dry form of eczema of the external ear, which may be a source of great discomfort to the patient from the continual itching within the ear, or from the accumulation of masses of desquamating epithelium. In the more acute cases perforation of the tympanic membrane may also occur, with secondary implication of the middle ear.

Otomycosis.—This is an inflammation of the external auditory canal due to the growth of a mould, of which the most common varieties are the *Aspergillus niger* and *A. flavus.* According to Highet, it is commoner in the tropics than in this country, and Dr. Galloway states that in the Malay Archipelago 70 per cent of all cases of ear disease are due to a fungus allied to the *Mucor mucedo.* It rarely occurs as a primary disease, but it is usually grafted on other conditions which Maurin describes as membranous, ceruminous, otorrhagic, eczematous, or cholesteatomatous. Its growth is favoured by moisture and by oily and watery solutions. The symptoms are usually pruritus or those of obstruction within the ear, but in the more acute forms there may also be considerable pain. The signs are those of inflammation of the external ear, but in addition a deposit may be seen dotted with black or yellow spots which, on removal, may cause bleeding and leave behind a raw surface. The diagnosis is confirmed by microscopical examination. The persistence of otomycosis depends largely on its association with the pus-producing micro-organisms and in consequence it is often very difficult to cure. The most successful applications are solutions of perchloride of mercury, tincture of iodine, nitrate of silver, or an alcoholic solution of boric acid.

Diphtheria of the External Meatus is seldom primary. It usually occurs as the result of diphtheritic infection from the pharynx or middle ear. When secondary, the condition may be overlooked owing to the general symptoms. As a primary condition, Lewin describes four cases in which the formation of a membrane on the concha and walls of the auditory canal was accompanied by much pain and fever. Frequently there is, in addition, considerable enlargement of the pre-auricular and cervical glands, as in a case recorded by Tommasi in which the diphtheritic infection was introduced by scratching the ear with a pen-holder which belonged to a girl with the same disease. Diphtheria of the auricle and external meatus may in rare instances be secondary to the throat, without implication of the middle-ear, from accidental infection with the oral secretions. The diagnosis is based on the general symptoms, on microscopical examination, and on the growth of cultures taken from the membrane. In addition to the ordinary constitutional treatment for diphtheria, frequent ear-baths of a warm solution of hydrogen peroxide tend to loosen the membrane. After the membrane has been removed healing of the raw patches may be hastened by the occasional instillation of drops consisting of a dilute solution of biniodide of mercury in rectified spirit.

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DISEASES OF THE TYMPANIC MEMBRANE

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It is very doubtful, and in fact most authorities deny, that any pathological condition of the tympanic membrane can occur, except as secondary to some affection of the external or middle ear; nor is this unreasonable, seeing that the outer epidermic layer of the drum is continuous with the epithelial lining of the external auditory canal, whilst the mucous membrane covering its inner surface is continuous with that of the tympanic cavity.

In many cases an apparently primary inflammation of the tympanic membrane will be found, on careful examination, to be really due to some previous inflammation of the middle or external ear, the obvious symptoms of which have subsided and have already been forgotten by the patient. Occasionally in the routine examination of an ear, thickenings or atrophic patches, or calcification of the drum, may be seen, but these again are the result of previous middle-ear trouble or suppuration. Nevertheless, from a clinical point of view, certain conditions may be described under the heading of diseases of the tympanic membrane.

Myringitis (inflammation of the tympanic membrane) without implication of the external or middle ear is said to occur as an acute condition from sudden cold in the ear, either from sitting in a draught, from a cold wind blowing in the ear, or in consequence of sea-bathing; from extensive heat, the result of scalding or of cauterising the external meatus by the galvano-cautery; from the instillation into the meatus of some chemical irritant; or from direct injury of the surface of the drum, as may occur in scratching the ear with a hairpin.

The symptoms are very similar to those occurring in acute inflammation of the middle ear, except that the hearing power remains good. The onset is usually sudden, and is accompanied by acute earache, frequently paroxysmal or neuralgic in character. The appearance of the drum varies; if the inflammation be slight, there may be merely local congestion of the affected part, but usually the whole drum surface is implicated with the formation of blebs and bullae, the result of serous or haemorrhagic exudation between its outer and middle layers. In the more acute cases the pain, though agonising, lasts perhaps for a few hours only, and then suddenly ceases on rupture of the bullae, which is accompanied by a sensation of something bursting in the ear and a slight sanious discharge from the meatus. In the less acute cases there is gradual absorption of the exudation with diminution of the pain. In rare cases the contents of the bullae are purulent, and in this connexion Grunert mentions a case of a primary interlamellar abscess containing numerous streptococci. As a result of acute middle-ear inflammation of influenzal origin, haemorrhagic blebs are frequently seen on the surface of the membrane and adjoining walls of the auditory canal, a condition which may occasionally be mistaken for primary myringitis.

Chronic inflammation of the drum may possibly be a sequel of the acute condition, but is more frequently secondary to some inflammatory affection of the external meatus which has already subsided. There are frequently no symptoms, with the exception of slight itching in the ear, or occasional fetid discharge from the meatus. As a result of desquamation of its epithelium, the surface of the drum may appear irregular and thickened and of a greyish colour; or crusts and inspissated pus may hide it from view; or it may be covered with granulations, sometimes of a large size.

Diagnosis is of importance from the point of view of prognosis, for if the disease be limited to the external ear, the possibility of any serious sequels, such as may occur in the course of a middle-ear suppuration, can at once be excluded. In the acute stage the diagnosis must be made by inspection, and, if absolutely necessary, by gently using a Siegle's pneumatic speculum. Inflation of the ear, to determine whether there is any fluid in the tympanic cavity, is not permissible, as it is strictly contra-indicated in acute inflammation of the middle ear. In the chronic stage, however, this may be done. When a perforation exists, the characteristic "perforation-sound," usually accompanied by crackling or bubbling râles, will be heard during the act of inflation of the ear; and on employing a Siegle's speculum, secretion may be seen to exude from the perforation. If, however, the middle ear be not affected, this will not take place, and the drum will be found to be intact. Also, in myringitis, impairment of hearing, if present, is very slight, whereas in middle-ear inflammation it is usually well marked.

The treatment depends on the condition found. In the earlier stages, before the bullae have burst, it is palliative only. It is sufficient to instil some sedative drops, and to protect the ear with warm, dry compresses of wool. Syringing should be avoided. If the pain does not diminish and the bullae increase in size, or if there be any sign that their contents are becoming purulent, they should be freely incised with a paracentesis knife, care being taken not to pierce the inner layers of the tympanic membrane. The ear should then be gently syringed out with a mild antiseptic solution, dried, and a small quantity of boracic powder insufflated, a strip of gauze being inserted as a drain. It may be necessary to change the dressings daily for a few days until the discharge ceases. In the chronic condition considerable patience may be necessary before a cure is obtained, chiefly on account of the difficulty of keeping the ear aseptic and of getting rid of the desquamating masses of epithelium. Treatment, therefore, should first consist in frequent instillations of an alkaline solution containing sodium bicarbonate, which will tend to soften and loosen the epithelial masses. After they have been removed the treatment is very similar to that already described for chronic diffuse inflammation of the auditory canal (*vide* p. 405). In the acute stage the condition may have to be distinguished from an acute middle-ear inflammation. In chronic myringitis, especially if the surface of the membrane be covered with granulations, it may at first be difficult to decide whether a perforation of the membrane exists or not.

For the sake of completeness the following **pathological conditions** of the tympanic membrane, although rarely seen, may be briefly mentioned. As a result of injury, usually after paracentesis, there may be a local heaping up of epithelium on the surface of the drum with the production of white, pearl-like bodies. *Herpes* of the tympanic membrane may also occur as a primary condition, but more frequently it is associated with herpes of the auditory canal and auricle. The only symptom may be neuralgic pain within the ear. On examination two or three

PLATE

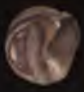
Fig. 1

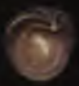
Fig. 2

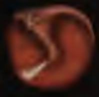
Fig. 3

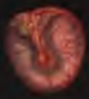
Fig. 4

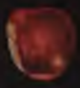
Fig. 5

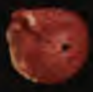
Fig. 6

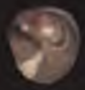
Fig. 7

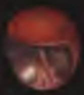
Fig. 8

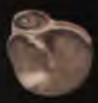
Fig. 9

PLATE XIII

DISEASES OF THE TYMPANIC MEMBRANE

Mr. Hunter F. Tod

Fig. 1.—Traumatic perforation of the tympanic membrane caused by an explosion. The eversion of the anterior lip of the perforation was due to violent blowing of the nose after the accident. (Dr. J. Dundas Grant.)

ACUTE INFLAMMATION OF THE MIDDLE EAR

Dr. J. Dundas Grant

Fig. 2.—Indrawn membrane.

Fig. 3.—Coppery tint and lustre of tympanic membrane seen at the earliest stage of inflammation of the middle ear.

Fig. 4.—Congested membrane.

Fig. 5.—Acute inflammation of the middle ear. Tympanic membrane red and bulging.

Fig. 6.—Acute inflammation. Black appearance of small perforation.

Fig. 7.—Cicatrix marking the position of the perforation in the case shewn in Fig. 5.

Fig. 8.—Bulging in the region of the "attic" in acute suppurative inflammation of that part.

Fig. 9.—Sunken cicatrix, following acute "attic" suppuration in the case shewn in Fig. 7.

vesicles, surrounded by a zone of congestion, may be seen on the membrane; they very quickly burst and leave behind tiny ulcers.

Vascular growths, or varices of the tympanic membrane, have also been recorded; if limited, they need not be interfered with, but, if necessary, can be cauterised under a local anaesthetic.

Tuberculous nodules have also been observed as sharply defined yellow or yellowish-red points, leading to multiple perforations, which eventually coalesce and cause considerable destruction of the drum membrane. This condition, though apparently primary, is usually associated with tuberculosis of the middle ear.

Traumatic Perforation of the Tympanic Membrane.—*Causation.*—This may occur from various causes, which are usually accidental, except in the case of imbeciles and children, who may deliberately poke an instrument into the ear; but in countries where there is conscription, intentional injury to the ear in order to avoid military service is not unknown. One of the most common causes of traumatic perforation is accidental injury to the drum from an unskilled attempt to extract a foreign body from the ear; but it may also occur, in spite of the greatest care, during the course of an operation on the auditory canal, such as the removal of exostoses. Another cause is a sudden and marked change in the atmospheric pressure, which must be very great in order to rupture a normal membrane. There is no doubt that in the majority of such cases rupture of the membrane occurs at the site of an old atrophic patch or scar. As examples of sudden increased pressure on the outer surface of the drum may be mentioned the firing off of a large cannon, a loud explosion (Fig. 1, Plate XIII.), or a blow on the ear with the hand. Similarly, suddenly diminished pressure will have the same effect, such as suction applied to the meatus, usually as a therapeutical measure. Less frequently, a rupture of the tympanic membrane may be caused by increased pressure from within, as in too forcible inflation of the ear by means of the catheter or Politzer's method; or it may accompany fracture of the base of the skull implicating the middle ear and roof of the external meatus. Traumatic perforation of the drum from a stroke of lightning has been recorded in very rare cases.

Symptoms.—When rupture takes place through a scar or atrophic portion of the membrane, the symptoms may be very slight. As a rule, however, at the moment of the injury, a loud noise or crack is heard in the ear, and the patient for some time afterwards is extremely giddy, and may feel faint and suffer considerable pain; not infrequently there is marked deafness associated with tinnitus. If the rupture be due to concussion, an elliptical perforation is usually seen in the inferior part of the membrane radiating towards its circumference. Its edges are generally coated with blood-crusts, and there may be ecchymoses and congestion of the tympanic membrane in its immediate neighbourhood. Through the gaping of the wound, the promontory of the inner wall of the tympanic cavity is seen to be of its normal yellowish-white colour. When due to direct violence, the perforation may be of any size or shape, depending on the instrument used and the violence employed. In addition to these local symptoms, it is important to determine to what extent concussion has affected the auditory apparatus. If the labyrinth be affected, tuning-fork tests will shew diminution of bone conduction and impairment of hearing for the high tuning-forks on the affected side. If, however, the injury be limited to the structures of the middle ear, and the labyrinth is intact, bone conduction will remain good, and the high tuning-forks will be well heard.

Diagnosis.—Traumatic rupture of the tympanic membrane is of considerable importance from a medico-legal point of view. The diagnosis of such a condition can only be absolutely certain when the case is seen within the first few days after the injury. The position of the perforation, its appearance, and the lack of all other signs of middle-ear inflammation or suppuration, are so characteristic as to be unmistakable. If, however, healing of the membrane has already taken place, or if, on the other hand, suppuration of the tympanic cavity has occurred before the patient first comes under observation, it may be quite impossible to determine whether a perforation really existed or whether the middle-ear suppuration is the result of the alleged injury or not. In a medico-legal case it is wiser not to give a definite opinion with regard to prognosis in the first instance, but to delay doing so until the patient is seen again after one or two months' interval. It is also very difficult to determine to what extent an alleged injury to the drum, which is said to have happened some weeks or months previously, is responsible for symptoms such as deafness, vertigo, or middle-ear suppuration, of which the patient afterwards complains. If, however, scarring of the membrane or calcareous deposits, pointing to an old middle-ear suppuration, are discovered on examination of the ear, too much importance should not be attached to the statements of the patient. If, on the other hand, there is no evidence of a pre-existing middle-ear inflammation and the appearance of the membrane is confirmatory, the probability of the patient's statement being correct may be accepted.

Prognosis.—In the simpler cases, as in rupture of the membrane from too energetic inflation of the middle ear, the prognosis is good. In the milder forms of concussion, in which the internal ear is not affected and the subjective symptoms quickly begin to subside, recovery can usually be guaranteed. In the more severe forms of concussion, especially if the deafness be very marked and due to implication of the labyrinth, the prognosis must always be guarded, as it is extremely difficult to say what improvement will take place. If middle-ear suppuration occur the result depends largely on the injury to the middle ear and to the virulence of the infection.

Treatment.—The ear must not be syringed, but should simply be protected by a dressing. On account of the giddiness, noises in the head, and general shock, and in order to avoid exposure and cold, rest in bed, even if not apparently necessary, is advisable. In rupture due to concussion the perforation, as a rule, heals quickly. If, however, it is due to direct injury from an instrument, middle-ear suppuration frequently follows, and must then be treated by the ordinary methods. In the more severe cases in which the inner ear is implicated it is essential that strict quiet should be enjoined; counter-irritation in the form of leeches or blisters may be applied over the mastoid process. When the noises and giddiness are intense sedatives may be administered. If deafness persist and is apparently due to implication of the nerve apparatus, strychnine may be given, $\frac{1}{60}$th grain three times a day, increasing the dose up to $\frac{1}{30}$th grain. If the tinnitus continue applications of the constant current may be tried.

Hunter F. Tod.

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H. F. T.

ACUTE INFLAMMATION OF THE MIDDLE EAR

By Dundas Grant, M.D.

The middle-ear system, including the Eustachian tube, the mastoid antrum and cells, and the tympanic membrane, is subject to inflammatory changes of various degrees of violence. The clinical characteristics vary according to the intensity and according to the part of the system mainly attacked. These affections will be considered as follows, the most generally accepted nomenclature being adopted:—

Acute Inflammation *of the Eustachian Tube*—Catarrh, Inflammation. *Of the Middle Ear Proper*—Catarrh (*vide* Otitis Media Serosa, p. 502), Inflammation, Non-perforative, Perforative. *Of the Attic. Of the Antrum and Mastoid Cells.*

The distinction between these cannot be made exactly in every instance, but most of the cases seen in practice fall under one of these headings, and it is obvious that they are sometimes merely successive stages of the same condition. The point at which a catarrh merges into an inflammation cannot be defined, and indeed the distinction depends more upon a clinical convention than upon an essential pathological difference. We shall assume "catarrh" to mean clinically a mild inflammation of the mucous membrane, unaccompanied by constitutional reaction or the formation of pus. Bacteriologically the difference probably depends on the presence or absence of pyogenetic microbes, or at all events on differences in virulence due either to the nature of the microbe or to the host's power of resistance. The intensity of the inflammation seen in the subjects of the exanthematous fevers is a familiar illustration of the latter. Limitation of the inflammation to one section of the middle-ear tract is theoretically almost impossible, but it occurs relatively, if not absolutely, in a sufficient number of cases to make its acceptance of considerable value in practice, the clinical pictures being often exceedingly distinct.

Acute Catarrh of the Eustachian Tube.—As this is not a fatal disease histological examination is not so available as in the more severe inflammations. This condition is invariably the result of an acute catarrh of the naso-pharynx and is particularly apt to occur in the subjects of hypertrophy of the adenoid tissue or of the various forms of rhinitis.

Hyperaemia and swelling of the mucous membrane, increased secretion of mucus and serum, and desquamation of epithelial cells are doubtless present. From the consequent narrowing of the tube the ventilation of the tympanum is interfered with and the contained air is absorbed. The excess of pressure of the atmosphere on the outer surface of the tympanic membrane, also described as a negative pressure in the interior, produces an "indrawing" of the membrane, the mobility of which is thereby impaired. This impairment of mobility diminishes the excursions of the ossicular chain, leaving the stapes abnormally deep in the fenestra ovalis.

The *symptoms* are dulness of hearing, fulness in the ears and dull buzzing in the head, coming on without any marked pain during the course of a nasal or naso-pharyngeal catarrh. The dulness of hearing is of the form characteristic of an obstructive lesion, on auscultation during inflation the sound characteristic of Eustachian narrowing is heard, whilst the hearing power is markedly improved after the inflation. The indrawn membrane (Fig. 2, Plate XIII.) is characterised by the prominence of the short process and the anterior and posterior folds, and the handle of the malleus is foreshortened and drawn up close to the posterior fold. Shrapnell's membrane is generally drawn in on to the neck and head of the malleus so as to make its outlines distinct, and presents light reflexes at various points. By posterior rhinoscopy the pharyngeal orifice of the tube shews more or less redness and swelling of the lip of the tube and white mucous plugs in the orifice. In the milder cases the mucous membrane of the tube returns to its normal condition, but in severe or neglected forms it is apt to undergo changes which result in tubal stenosis and chronic abnormalities in the tympanum.

The *treatment* is mainly that appropriate in nasal catarrh, but in addition some form of inflation of the tube and tympanum is required. As a rule Politzer's method is sufficient, and the Eustachian catheter is necessary in exceptional cases only. In such cases it is well to introduce into the catheter a few drops of liquid vaseline-oil containing a trace of menthol (gr. i ad ℥i). This may be repeated on the following day and at increasing intervals, during which the patient may practise some form of self-inflation. This may be effected by means of Valsalva's method (*vide* p. 366). The mouth and nose are previously filled with vapour by the inhalation of steam from a pint of water at a temperature of 120° F. This may be medicated by the addition of a trace of menthol (gr. ¼-½), 1 minim of chloroform, and 1 dram of compound tincture of benzoin; the presence of the chloroform facilitates the passage of the vapour up the Eustachian tubes. It is undesirable that the patient should be exposed to the cold air immediately after the use of this steam inhalation, and when this is unavoidable Valsalva's method of inflation without vapour may be practised, or a simple self-inflator of the following description may be employed. It consists of an india-rubber tube of large calibre and about 12 inches in length, having at one end a glass tube covered by an infant's-bottle teat and at the other a glass mouthpiece in which a small loose pellet of cotton-wool is placed. The mouthpiece may either be expanded in trumpet-form so as to be placed over the lips, or it may be compressed so as to be introduced into the mouth. Five drops of such a combination as the following should be placed on the wool each time of use:—Menthol, gr. xii; chloroform, ℥ss; acetic ether and rectified spirit āā ʒii. The nose-piece is then introduced into the freer of the two nostrils, pointing backwards rather than upwards, the ala nasi is compressed over it and the opposite nostril is closed, the mouthpiece is then adapted to the mouth, and the patient blows through it in such a way that he distends his cheeks. The expired air, medicated with menthol and chloroform, is thus forcibly compressed in the naso-pharynx and driven up the Eustachian tubes with such freedom as their degree of patency permits. The least possible amount of force should be used compatible with inflation of the tympanum—"cracking the ears." It is undesirable that elderly people or the subjects of arterial disease should make these forcible expiratory efforts, and in them the inflation should be effected by Politzer's method.

When the acute stage subsides, but the narrowing of the tube is slow in disappearing, astringent and antiseptic applications may be made to the naso-pharynx by means of a wool-holder so bent as to be passed easily behind the soft palate. For this purpose one of the best applications is a solution of chloride of zinc (gr. xv ad ʒi). This should be followed by inflation. A few drops of a solution of the same salt (gr. v ad ʒi) may be introduced into the catheter and blown into the pharyngeal end of the Eustachian tube in the form of a coarse spray.

Acute inflammation of the Eustachian tube of an intense character results from the severe inflammatory conditions in the naso-pharynx found in diphtheria, erysipelas, and the various infectious diseases. Extension to the middle ear with the symptoms of perforative inflammation is inevitable, but the occurrence of violent pain shooting up to the ear (apart from that occasioned by inflammation of the lymphatic glands) and extreme dulness of hearing would indicate the threatening of such an extension. Obviously such tests as auscultation during inflation of the tympanum through the Eustachian tube are quite impracticable. Small follicular abscesses which lead to the formation of ulcers have been described (Schwartze, Walb, (12)) in the mouth of the Eustachian tube, especially in the subjects of adenoid vegetations.

The *treatment* must be confined to that appropriate to the general systemic infection and to the naso-pharyngeal condition.

Acute Tympanic Catarrh (*vide* Serous Otitis Media, p. 502).

Acute Inflammation of the Middle Ear.—This heading includes those forms of inflammation which differ from the simple catarrh in the degree of the reaction; this depends, no doubt, upon the presence of toxins or pyogenetic organisms in a favourably disposed soil. Our classification is an arbitrary one, founded on clinical data and possessing some clinical convenience, but, in reality, the various forms to be described are degrees or stages of the same disease.

Acute inflammation of the tympanum is a convenient name for a certain number of cases presenting acute symptoms, such as occur in the earliest stages of purulent otitis, which, however, subside without any evidence, by spontaneous evacuation, of suppuration. There is, no doubt, an invasion of pyogenetic organisms, but these are either in small number or of slight virulence, or the bactericidal power of the patient's blood is equal to their destruction.

The *causes* are mainly exposure to cold, favoured by the presence of inflammatory conditions in the nose and naso-pharynx, and especially such general infections as measles, scarlet and enteric fever, diphtheria, pneumonia, and influenza.

The *pathological conditions* are hyperaemia and infiltration of the mucous membrane with superficial erosion and a sero-mucous exudation containing a little pus and sometimes a trace of blood, with columnar epithelial debris and micro-organisms of various degrees of virulence.

The *symptoms* are a throbbing pain in the ear supervening on a feeling of malaise, buzzing noises in the head, and a slight degree of dulness of hearing. There are two very characteristic appearances of the tympanic membrane, one of which may be called the "coppery" and the other the "congestive." While the outer layer of the tympanic membrane retains its normal shine and transparency, and while the deeper layer is congested, the membrane presents a peculiar metallic appearance like the surface of a brightly-polished copper kettle. This is not often seen, because the extension of the inflammation to the outer layer soon produces a loss of the lustre and transparency on which the appearance depends (Fig. 3, Plate XIII.). The congested membrane is recognised by injection of the vessels round the periphery, and further by the prominence of the vessels running approximately down the posterior aspect of the handle of the malleus (Fig. 4, Plate XIII.). The rest of the membrane is of a pale pinkish tinge, devoid of lustre or translucency.

The symptoms usually develop with considerable rapidity, the pains fluctuating in their severity. The temperature in adults in mild cases may not rise above 100° F., but in children it may be much higher, and from the occurrence of delirium and convulsions meningitis may be suggested. The dulness of hearing is very considerable, and presents the characters of an obstructive deafness, although from accompanying congestion of the labyrinth bone-conduction may be diminished.

Prognosis.—In non-perforative cases the acute symptoms should subside in about three days. Hearing is generally completely restored, especially under judicious treatment, but sometimes localised adhesions remain and interfere with function.

The *diagnosis* must be made from the various diseases of the ear characterised by pain, such as neuralgia, furuncle, and myringitis. In neuralgia there is the absence of deafness, fever, and visible change in the membrane, and a reflex cause, such as a carious tooth, is usually discoverable. In furuncle of the meatus the pain is more distinctly paroxysmal, the dulness of hearing only supervenes when the swelling increases so as to produce mechanical atresia of the meatus—although it has been noted that a degree of nerve-deafness sometimes accompanies it — and the localised swelling of the meatus with extreme tenderness on pulling the tragus is usually obvious. Acute myringitis is characterised by the comparative absence of dulness of hearing which contrasts most strongly with the high degree of change in the appearance of the membrane. There is also absence of narrowing of the Eustachian tube.

Treatment.—Apart from the general treatment of the inflammatory condition, such as rest in bed, liquid diet, the avoidance of tobacco, alcohol, and heating drinks, some local measures are of considerable value. Among the most widely used is the instillation of glycerin, containing 5 or 10 per cent of carbolic acid. This should be prepared from pure phenol and neutral glycerin, the solution being very thoroughly made so as to obviate the possibility of any undissolved droplets of the phenol irritating the skin or tympanic membrane. These drops act as a sedative and antiseptic, and they probably also exercise an exosmotic effect. The addition of 25 per cent of alcohol may insure the solution of the phenol and increase the antiseptic effect. Cocaine, adrenalin, and atropine in combination may be introduced in the form of drops, or the "ovoids" devised by Mr. R. H. Woods (15) may be introduced. These consist of small radish-shaped cones of gelato-glycerin, each containing of liquid extract of opium gr. $\frac{1}{7}$, hydrochloride of cocaine and sulphate of atropine of each gr. $\frac{1}{14}$, with 2 per cent of creolin. They may be introduced every six hours till the pain is subdued, but have to be syringed out. Mr. Tod recommends the following formula for "drops": cocainae, gr. ii.; morphinae, gr. i.; acid. carbolic., gr. iii.; ol. amygdal. dulc., ʒi.

The application of cold by means of the cold-coil or an ice-bag is often of value in reducing the inflammation and preventing extension to the adnexa. Aural ice-bags are procurable, which are like small india-rubber hot-water bottles, with a hole through which the auricle can pass. There are, however, many cases in which cold does not relieve, and the patient derives comfort from heat, as afforded by means of warm water instead of cold in the coil, or the application of warm fomentations well wrung-out and covered with oil-silk or gutta-percha tissue. These may be moistened with simple warm water, boracic solution, a 3 per cent solution of acetate of aluminium, or spirit of wine. The latter has been recommended as not so likely as watery fluids to cause maceration of the cutis. If the mastoid is tender, leeches (natural or artificial) to the number of three or four may be applied, it being remembered, however, that the swelling which follows their suction may mask the advent of mastoid periostitis, and may have to be discounted.

Should the pain and fever not subside after about three days the membrane must be incised, as described in the section on purulent perforative otitis (p. 424). The exudation in the tympanum may be allowed to find its way out, or this may be hastened by the use of Siegle's exhausting speculum or Sondermann's suction apparatus. The antiseptic drops above described will have rendered the external meatus fairly aseptic. It will then be only necessary to keep a small strip of gauze very loosely in the meatus and an antiseptic dressing over the ear. If the discharge soaks through the gauze strip, it should be removed and a fresh one inserted, the meatus being wiped out with sterile absorbent wool. If the discharge becomes very profuse and purulent the treatment must be carried out as in acute perforative inflammation.

If subsidence of the pain occurs without suppuration, gentle inflation through the Eustachian tube must be practised with increasing intervals till normal hearing is restored or until no further improvement is obtained. The local treatment of the throat and nose must then be carried out; external massage of the neck from the mastoid process downwards during the act of deglutition is very useful, while appropriate treatment is instituted for any diseased condition in the nose or nasopharynx. The removal of post-nasal adenoid vegetations, when present, is the best means of preventing recurrence.

It occasionally happens, especially in children, that the effusion finds vent through the Eustachian tube, with subsidence of the symptoms following a discharge from the nose and throat, which may, however, escape observation. In other cases the infection may, from unfavourable anatomical or other conditions, extend into the mastoid cells and produce an acute mastoiditis without perforation of the tympanic membrane. The slightest suspicion of this course of events would call for instant and free incision of the membrane.

Acute Purulent Perforative Inflammation of the Middle Ear.—It is scarcely possible to conceive of a disease in which early diagnosis and treatment are more important than in acute purulent inflammation of the middle ear. The dangers incident to the condition itself are fairly well known.

It may be defined as a severe inflammation of the mucous lining of the cavities of the middle ear excited by the invasion of pyogenetic organisms, leading to the formation of pus and perforation of the tympanic membrane as the result of pressure and inflammatory necrosis of its substance.

Pathology.—The vascular and nutritive changes incident to the inflammation bring about a swelling of the mucous membrane with exudation of a fluid rich in globulins and containing a few epithelial cells and numerous polymorphonuclear leucocytes. The exudation rapidly becomes muco-purulent, and contains chiefly large mononuclear lymphocytes, whilst the surface of the swollen mucous membrane loses its polish, being eroded entirely or in part. If the virulence is at all considerable, the inflammation must extend to the periosteum, and in the most severe cases to the bone. The vessels of the tympanic membrane become dilated and thrombosed, so that the nutrition of its substance suffers, and it readily undergoes necrosis at its weakest part. A perforation having thus taken place, the microbes present in the external meatus obtain ready entrance and add increased virulence to the existing inflammation, giving rise to various kinds of products according to their degree of pathogenicity in relation to the resisting power of the patient's tissues. Increase in amount of the discharge takes place, and at the same time it loses its stringy character and becomes a milky pus.

The *bacteriology* of acute suppuration of the middle ear has been the subject of much investigation, though with somewhat varying results. The simple primary factor has hitherto generally been considered to be some form of diplococcus, and after perforation a mixed infection has resulted from the invasion of other bacteria, mainly staphylococci and streptococci. Dr. Wyatt Wingrave in 49 cases found diplococci 36 times (*Micrococcus catarrhalis* 20, pneumococcus 9, meningococcus 4, gonococcus 3); streptococci, 11; staphylococci and *Bacillus coli*, each 4; *Streptococcus mucosus*, *B. proteus vulgaris*, *B. tuberculosis*, *B. subtilis*, each twice; *Bacterium termo*, non-tuberculous acid-fast bacilli, spirochaetae, *B. influenzae*, and *Micrococcus tetragenus*, each once. According, however, to observations at the meeting of the German Otological Society in 1907 about 46 per cent are caused by the *Streptococcus pyogenes*, 18 by the *Diplococcus pneumoniae*, 10 by the *Streptococcus mucosus*, and 9 by the *Staphylococcus pyogenes aureus* and *albus*.

As regards the course of the disease in relation to the predominant form of bacteria there is found to be a probability that operation will be required in 1 out of every 3 with *Streptococcus pyogenes*, and 1 out of every 2 with *Streptococcus mucosus*, which is apt to invade the bone. The course appears to be protracted in the staphylococcal cases, but possibly the prolongation of any given case really admits of the supervention of the invasion by staphylococci whatever may have been the original bacterial factor. The pneumococcal cases are said by Zaufal to be characterised by a cyclical course, that is, to undergo a crisis. Körner (5) insists, however, that when this occurs the cause is really pneumonia, signs of which may be detected if the chest is carefully examined.

Etiology.—The causes of catarrhal or non-perforative inflammation of the middle ear must, before they can produce a perforative suppuration, be supplemented by some factors of a traumatic or pathological nature. Thus, among the chief causes are blows on the ear, the incidents of bathing, operations in the nose, plugging the posterior nares; whilst among the more purely pathological factors are the presence of eruptive or other fevers, or of some debilitating influence such as fatigue, privation, exposure to extreme cold, diabetes mellitus, tuberculosis, or other cachexia. The passage of foreign bodies up the Eustachian tube is an important cause; in addition to the grosser forms of foreign body, mention must be made of the entrance into the tubes of naso-pharyngeal secretions, and matter coughed or vomited up, especially when the tubal orifices are abnormally patent. Thus, in the subjects of exhausting diseases, such as enteric fever, or of the wasting incidental to old age, the walls of the tube are thinned by absorption of their fatty cushion and the canal is considerably widened. The conditions in infants are also favourable for the entrance of foreign bodies, the tubes being shorter, straighter, and wider in proportion than in the adult.

Symptoms.—The onset of this disease is usually marked by severe throbbing pain in the ear, often preceded by constitutional disturbance evidenced by chilliness, and, in extreme cases, by rigors or vomiting, fever, and rapid pulse. There is at the same time a fulness in the head, buzzing noise, and rapidly increasing deafness in the affected ear. In young children there may be rolling of the head, screaming, convulsions, squinting, and other symptoms of meningitis, or, at all events, of meningeal irritation, whilst in adults some amount of delirium and stupidity may be present. A high leucocytosis strongly suggests suppuration, but its absence does not prove the absence of pus.

On local examination we usually find the membrane at a stage when its normal features are obliterated by the swollen and protruding tissues. It then presents the appearance of a red swelling merging insensibly into the walls of the meatus, and having on its surface some flakes of desquamated epithelium. There may be a general bulging of the whole membrane, but more often it is mainly the posterior part which is seen. This depends on the inclination of the membrane, the posterior part lying in a plane somewhat external to the anterior, so that when it bulges it quite conceals the latter along with the malleus. This is seen in the coloured plates (Figs. 5 and 7, Plate XIII.). In earlier stages the membrane may present the "coppery" appearance of the one characterised by vascularity of the periphery, and of the malleal leash described above (Figs. 3 and 4, Plate XIII.). At an intermediate stage, if the swelling of the posterior part is not too prominent, the anterior part may bulge as well, the handle of the malleus being dimly distinguishable or more often only its short process. Sometimes the bulging is in the form of a nipple-shaped sac at the tip of which perforation takes place. The membrana flaccida or the postero-superior quadrant of the membrane are favourite sites for these projections, and the cases in which they occur are generally somewhat obstinate. A portion of the bulging becomes dull yellow and sodden, and gives way, discharging a mucopus more or less stained with blood. In some cases of unusual intensity, more especially those associated with influenza, the discharge may be preceded by slight haemorrhage, and the proportion of blood in the discharge is considerable. It may in some instances be so great as to give rise to the identification of a class known as *otitis media acuta haemorrhagica*.

Small perforations, if they can be sufficiently cleaned up, have a blackish appearance (Fig. 6, Plate XIII.), whereas large ones reveal the reddened mucous membrane of the inner tympanic wall. The cleansing cannot always be effected, and the presence of a perforation may be surmised by the visible pulsation of the overlying secretion, it being scarcely possible for this to be communicated to the fluid through an intact tympanic membrane. The so-called perforation-sound may be obtained on auscultation during inflation through the Eustachian tube. A perforation is often covered by what appears to be a succulent granulation, but is really the pouting of a circular ring of infiltrated mucous membrane. It may be snared or punched off when its annular nature is at once recognisable. The perforation-sound may, however, be unattainable on account of impermeability of the Eustachian tube, or because the tympanic cavity is completely obliterated by the swollen mucous membrane which may at the same time act as a valve closing the perforation and preventing the transmission of air. Again, in "attic" perforations the sound is often absent (*vide* p. 430). Opinions differ as to the most frequent position of perforations, and it has often been stated to be the antero-inferior part. In many cases, however, it is really the posterior part, but appears to be the anterior on account of the bulging or overhanging of the posterior. In severe cases, especially those occurring during infectious fevers, considerable destruction takes place, large reniform or heart-shaped perforations often remaining round the tip of the manubrium.

A certain amount of tenderness over the mastoid region, especially the apex, with moderate infiltration of the soft parts, is usual at the commencement of the inflammation. It ought to pass off on the occurrence of the discharge, and does not necessarily indicate any implication of the bone, as such symptoms coming on after a week would do.

In average cases the discharge begins to diminish in about a week, and the perforation heals up in from two to three weeks. In very exceptional cases the evacuation of the secretion is followed by almost immediate closure. It is more usual for the discharge to continue, though gradually diminishing, for three or four weeks. If by this time it continues copious and does not shew signs of diminishing, there is probably some unfavourable factor at work, such as incomplete drainage due to the extremely small size of the perforation, or its too high situation. Apart from this a very frequent cause of persistence is implication of the antrum or mastoid cells, which may be termed an empyema and may be present without any such external change in the mastoid as to lead the patient to complain of it. The former condition may be eliminated by enlargement of the perforation, as indeed may also the latter, but this as a rule calls for surgical opening of the mastoid cavities.

The *prognosis* turns in great measure on the original cause, whether infectious (exanthematic), "genuine" (catarrhal), or traumatic. In the "genuine" and traumatic cases, under almost any reasonable method of treatment, the general rule is for the inflammation to subside, the suppuration to diminish gradually, and for the perforation to close without any serious lowering of the hearing-power. In the infectious cases, on the contrary, there is a great tendency for the suppuration to persist, mixed infection from the invasion of external pathogenetic organisms to take place, and for the disease to extend to the deeper tissues and the remoter parts of the petrous bone. We thus get cario-necrotic changes in the bone with softening and breaking-down, and unification of the various cells in the petro-mastoid region. In a minority of instances extension to the labyrinth or to vital parts, such as the extradural space, the pia-arachnoid membrane (usually through the intermediary of the labyrinth), the lateral sinus, and more rarely the parenchyma of the brain or cerebellum, takes place. A few hints as to the detection of threatened extension in these various directions will be given in this section, in connexion with mastoid disease, but their more complete description will be found in the article on the dangerous sequels of suppurative inflammation of the middle ear (p. 475).

If a differential blood-count shews a rise in the percentage of polymorphonuclears too great in proportion to the increase in leucocytosis, the inference is that the degree of toxaemia is out of proportion to the systemic power of resistance, and the prognosis is therefore less favourable than in the opposite condition.

In some cases the suppuration may subside, but leave residua by which the hearing-power is impaired. Thus, the perforation may remain open, its edges having cicatrised, or it may become closed by a thin cicatrix, which if large and relaxed may interfere materially with hearing. Adhesions often bind the ossicles together. In other cases the acute symptoms settle down, but the discharge continues owing to causes detailed in the section on chronic suppuration of the middle ear.

Diagnosis.—In the stage before perforation has occurred the diagnosis is made on the same lines as in the non-perforative form. When perforation has taken place the disease must be distinguished from the others characterised by a discharge from the ear, especially external otitis and tuberculous otitis media, circumaural abscesses, and exacerbations of chronic suppurative otitis. From external otitis it is to be distinguished by the more copious discharge, which may be stringy from the presence of mucus, the early impairment of hearing, the lesser degree of swelling of the meatal walls, and the presence of a perforation.

In tuberculosis of the middle ear there is absence of pain, the dulness of hearing is extreme, and there is often more than one perforation. The discharge is usually thick and contains tubercle bacilli. Some of the various general evidences of tuberculosis may be present. Occasionally abscesses in the surrounding parts open into the membrano-cartilaginous part of the meatus—presumably through the fissures of Santorini—producing a copious purulent discharge. This is preceded by the external swelling, pressure on which increases the flow ; it is not ushered in by deafness, and there is no tympanic perforation. Such abscesses generally arise in the parotid tissues. Suppuration in mastoid cells sometimes leads to perforation of the posterior wall of the meatus and discharge of pus from the meatus. The fistula can be detected by means of a bent probe, and there is generally a history of former suppuration and evidence of mastoiditis. From acute exacerbations of chronic suppuration of the middle ear it can be distinguished by the absence of history of former deafness or discharge, and of changes such as the presence of cholesteatoma or callous-edged perforations, which only result from long-standing disease. Sometimes the diagnosis can only be made as the inflammation subsides, and sometimes it cannot be made at all.

The *treatment* varies with the age of the disease. In the absence of symptoms which indicate an inflammation of such severity as to lead to perforation, the treatment is the same as that prescribed for acute simple inflammation, as regards rest, diet, instillations, temperature-regulating coils, and especially leeching. To these may be added the administration of internal remedies such as aperients, for example calomel, narcotics and analgesics, sulphonal and phenacetin, bromide of potassium, and salicylate of sodium, but their use must not be allowed to lead to procrastination with regard to paracentesis.

If, therefore, there is pyrexia (over 100° F.), pain such as to destroy a night's sleep, and bulging of the tympanic membrane, paracentesis is at once called for. This is specially necessary when there is mastoid tenderness or swelling, or symptoms of meningeal irritation (frequent in children). If these symptoms present themselves even in a very mild degree in the course of an infectious fever, the indication is all the stronger.

The operation of paracentesis, or rather incision of the tympanic membrane, serves for the evacuation of the purulent contents of the cavity, for the depletion of the infiltrated mucous membrane, and for the restoration of the atmospheric pressure; it also favours the drainage through the Eustachian tube in the same way as the removal of the finger from the top of a filled pipette allows of the escape of the liquid. It is carried out by means of a fine knife which, whatever the shape of its blade (lancet-shaped, crescentic, or straight), must be double-edged. With it a cut of about a quarter of an inch in length is made in the most protruding portion of the membrane or in its posterior half. Owing to the inclination of the membrane it is easier to make a free opening by cutting from below upwards than from above downwards. If there be any tendency to swelling of the deeper part of the postero-superior wall of the meatus, the incision should be extended, or a fresh one made down to the bone of this part. Previous to this operation such sterilisation of the meatus as is practicable should be carried out. This cannot be ideally perfect, but it is wise to syringe out the meatus with a solution of 1 in 3000 of biniodide of mercury and to scrub the auricle and outer part of the meatus (membrano-cartilaginous) with Lister's strong solution (perchloride of mercury 1 in 4000 and carbolic acid 1 in 20), or with 1 in 3000 of biniodide of mercury in equal parts of rectified spirit and water. If antiseptic instillations have already been made, so much the better. The most convenient source of illumination is an electric head-light which can be used without much raising of the patient's head. Anaesthesia is often unnecessary, and the expert can "rush" the little operation with very slight tax on the patient's endurance, for which the subsequent relief usually atones. In very nervous cases, nitrous oxide or chloride of ethyl may be administered, but ordinarily it is sufficient to apply a few crystals of cocaine and adrenalin. Bonain's solution (*vide* p. 383) produces a fair local anaesthesia when applied carefully to the site of the incision, and Dr. Gray's solution favours penetration through the membrane, but, unfortunately, in a few instances rather alarming symptoms of aniline poisoning have been produced, though not in cases of intact membrane. After the incision the meatus should be gently dried out, and may be exhausted by means of Sondermann's apparatus or Siegle's speculum. A compress is then applied with or without a loose strip of gauze in the meatus. It is on the whole safer to omit the strip rather than risk its acting as a plug instead of a drain. If the discharge is very profuse it must be dried out very frequently and syringed occasionally. The surface of the cotton-wool mop may be sterilised by "toasting" over a lamp. In case of pain, drops of carbolised glycerin (1 in 20) should be instilled and an alcoholic compress or ice-bag applied. If the discharge does not shew signs of diminishing after the pain subsides, the ear may be syringed with a weak solution of formalin (1 in 500) and drops of boric acid (1 in 16) dissolved in rectified spirit (25 increased to 50 per cent) and water. When the discharge has become very slight, a little finely-powdered boracic acid may be insufflated. It will then be advisable to practise inflation of the middle ear by Politzer's method.

Among the less familiar methods of treatment is Bier's passive congestion (*vide* p. 387).

Suction is often of benefit and may be practised by means of Siegle's exhausting speculum, or a tube inserted into the meatus while air is drawn out by means of a syringe or the mouth, but better still by means of the less well-known suction apparatus devised by Sondermann.

In obstinate cases, especially those in which operation is refused, it is advisable to try the effect of a vaccin prepared from cultures made from the discharge. This is more likely to succeed in cases due to a pure infection than in those due to a mixed one.

Acute Otitis Secondary to Infectious Diseases.—The secondary inflammations of the middle ear are most common in connexion with scarlet fever, diphtheria, measles, influenza, enteric fever, and pneumonia, as in these the naso-pharynx is always more or less implicated; the most probable mode of infection is through the Eustachian tube (salpingogenous) as there are reasons for believing that infection through the blood-current (haematogenous) is improbable. Thus, in some of Kobrak's (6) examinations in which bacteria were found in the blood, the organisms in the ear were of a different species. In infectious diseases of known bacteriology, such as diphtheria and enteric fever, the specific organism has occasionally been found in the early stages, but in the later stages the predominant bacterium is practically always the *Streptococcus pyogenes.* In the early stages, the effusion obtained by paracentesis is often sterile, and we are driven to the conclusion that the inflammation of the middle ear may be due to the toxins apart from the bacteria. The streptococci no doubt find their way from their usual habitat—the cavities of the mouth and nose—either through the tube or, as has been suggested, through the lymphatics of the tonsils.

Recent investigations (6) in regard to the *bacteriology* of the various "secondary" forms of otitis are briefly as follows:—In scarlet fever during the first few days the contents of the tympanum were found sterile in spite of the presence of acute inflammation, whilst later there was invariably a pure culture of *Streptococcus pyogenes.* Diphtherial cases varied and in several there was sterility, in others pneumococcus, the *Bacillus pyocyaneus* or the *Micrococcus candidans*, but in none the Klebs-Löffler bacillus. In the published cases in which the diphtheria bacillus is believed to have been found, there is reason to believe that the virulence was not tested, and that the organism was merely the pseudo-diphtheria bacillus. It is probable that the acute otitis occurring in diphtheria is often of the non-specific form such as may occur in any debilitated subject (Brieger (6)). The otitis of measles was at first found to give sterile results, but after a week there is almost invariably positive evidence of *Streptococcus pyogenes*, very seldom of pneumococci. In influenza the specific bacillus has been found (after much search) by some observers (Scheibe (6)), but not by others (Kümmel), the *Streptococcus pyogenes* being almost invariably the active bacterium. In a few cases the *Streptococcus mucosus* and other micro-organisms were present. The *Micrococcus intracellularis* has been detected in the aural discharge in epidemic cerebrospinal meningitis, but further investigations in the light of more recent studies of the various forms of streptococci are desirable. The *Bacillus typhosus* was apparently the sole cause in one of the comparatively few observations of typhoid otitis which have been made, but as a rule the bacteria found have been those characteristic of "primary" otitis, namely streptococci, staphylococci, pneumococci.

The *clinical features* of these "secondary" or "concomitant" forms of suppurative otitis are in general those of the "primary" or "genuine," but as a rule are more severe. The cachectic condition of the patient and the difficulties in the way of early diagnosis and treatment account for the less favourable course.

In scarlet fever, which accounts for 2·8 per cent of Bezold's cases of acute suppuration of the middle ear, the acute otitis usually arises at the height of the fever or at the stage of desquamation, and is most severe in those cases in which the naso-pharyngeal inflammation takes on a pseudo-diphtheritic form, apart from the coincidental occurrence of true diphtheria. When its occurrence is delayed till the stage of desquamation, the otitis is of a much less severe form. It is usually ushered in by severe pain in the ear, increase of fever, and nervous disturbance such as delirium or somnolence. Dulness of hearing is usually very marked. The cervical glands are generally much swollen. It often happens, however, that no pain is complained of, and the first sign of otitis is the occurrence of the discharge. In many cases the inflammation subsides without going on to perforation, and even when perforation takes place, rapid healing frequently follows, but often—especially in the diphtheroid forms—destruction of a large part or even the whole of the tympanic membrane rapidly ensues. The lining of the tympanum is often so swollen as to fill up the opening, and to look almost as if the membrana tympani were still present. The suppuration is apt to be obstinate and to last two or three months, leaving often a persistent perforation or sometimes a chronic suppuration, with its various evil effects and dangers.

The local treatment by means of carbolised glycerin or Woods's aural suppositories (ovoids) is indicated in the early stages, and if pain, fever, and bulging of the membrane are present, paracentesis should, if practicable, be performed. When discharge has set in, the ear should be irrigated with simple warm boiled water or a weak borax lotion, and be loosely covered with a pad of sterilised or antiseptic gauze. The further treatment is the same as in the non-infectious cases, both as regards conservative and operative measures. Mr. Woods (16) advises, as a prophylactic measure, that in all cases of scarlet fever or measles the ears should be cleansed by syringing with creolin lotion. If the ears then suppurate they are syringed carefully and frequently, and filled once daily with a saturated solution of boric acid in 50 per cent spirit.

Diphtheria may complicate scarlet fever at any stage, but most frequently during convalescence. It is then very apt to cause the most severe otitis. Extensive perforation of the membrane takes place and through it the fibrinous exudation may be recognised. In addition to the local measures already detailed, the general treatment appropriate for diphtheria should be carried out, including injections of antidiphtherial and antistreptococcic serums. The usual symptoms of purulent otitis are present, and when perforation takes place there may be at first very little discharge, and no appearance of diphtheritic exudation in the tympanum, though the latter may develop later on.

Measles causes purulent inflammation of the middle ear more frequently than is generally supposed; Bezold finds that 5·7 per cent of his cases of purulent otitis are due to this disease. It has been noted that the upper part of the Eustachian tube is often normal, while the naso-pharynx and tympanum are both in a purulent condition. Probably the aural condition is due in the first instance to the action of the toxins of measles, which render the mucous membrane a suitable nidus for the streptococcus.

The otitis media of enteric fever is not a common occurrence (0·2 per cent of cases of acute otitis—Bezold). It usually occurs in the later weeks of the disease, and runs a course differing in no particular respect from an ordinary primary otitis. Its pathogenesis has been variously explained, but in great probability it is due to extension or invasion through the Eustachian tube, the lumen of which is widened owing to the cachectic thinning of its walls, while at the same time its protective ciliary mechanism is weakened. The naso-pharynx supplies the typhoid bacilli which, as has been already stated, may be found actually in the middle ear. It should be noted that the dulness of hearing occurring in enteric is often due to an implication of the labyrinth and still more often to a blunting of the central nervous centres, quite independent of any change in the middle ear (*vide* Vol. I. p. 1130).

Influenza, during epidemic years at least, causes a large number of purulent inflammations of the middle ear, 6·3 per cent of Bezold's cases being due to this. Although frequently identical with primary otitis the influenzal form is characterised by the frequent occurrence of haemorrhagic bullae on the membrane, their rupture being accompanied by slight bleeding from the ear (*vide* p. 409). There is, in fact, a myringo-tympanitis. The tendency to implication of the mastoid cells is considerable, and the mastoid operation is often required. The deafness sometimes induced by influenza is often due to inflammation of the labyrinth or of the auditory nerve.

In the *treatment* of the purulent otitis media arising in the course of these diseases, the usual principles apply, but paracentesis should be performed on even less indication than in the primary cases, and the cleansing of the naso-pharynx is still more indispensable if the patient's condition does not render its carrying out impossible. Prophylactic nasal irrigation is in these cases more likely to induce than to prevent aural suppuration, but a few drops of liquid vaseline, containing in each ounce 3 grains of menthol and 5 minims of oil of eucalyptus, may occasionally be poured into each nostril while the patient is lying on his back.

Acute inflammation of the middle ear in infants presents some important features, among which may be noted the frequent association with bronchopneumonic and alimentary disturbances, the delay in spontaneous rupture of the tympanic membrane, and the presence of symptoms simulating meningitis.

In the association with pulmonary and alimentary disturbance, the ear disease may be either cause or effect. The swallowing or inhalation of the aural pus may lead to infection of the alimentary or respiratory mucous membrane, whereas, on the other hand, infective materials vomited or coughed up may readily pass through the short and relatively wide Eustachian tube of the child. The meningitic symptoms can be explained by the persistence in a modified form of the embryonic petrosquamosal cleft by which the tympanum and the meninges approximate to a degree which is not found in the adult, and, furthermore, by the characteristic excitability of the nervous centres in the child under the influence of any peripheral irritation, but especially of one in such close proximity. It is well known that the symptoms of apparently fatal meningitis often disappear on the occurrence of a discharge from the ear.

Two forms are to be distinguished, the otitis of the new-born and that of the young child. The former is due to the entrance of amniotic fluid and various foreign bodies, such as hair and meconium, and has a tendency to subside without disturbance of health. In two reported cases, however, the gonococcus was found. The otitis of the infant or young child is a more dangerous condition, and one of considerable frequency. Hartmann found, in the Hospital for Infectious Diseases in Berlin, that 78 per cent of the infants were the subjects of otitis, and Kessel found that, of 108 of the children under one year old who died in that institution, 85 had middle-ear disease as shewn by autopsies. The bacterial factors appear to be very various, Kessel having found in 38 of the cases above referred to, pseudo-influenzal bacilli in 19, pneumococci in 10, streptococci in 4, staphylococci in 2, the *Bacillus pyocyaneus* in 1, and Friedländer's pneumobacillus in 2. Preysing's more recent investigations in 154 cases with purulent, mucous, or serous fluid in the tympanum, shewed: Pure culture of pneumococci in 96, pneumococci and saprophytes in 13, pneumococci and staphylococci in 3, pure streptococci in 1, *Staphylococcus pyogenes aureus* in 3, staphylococci and saprophytes in 2, saprophytes alone in 3, and sterility in 33. The condition is, therefore, in by far the majority of cases, pneumococcal.

The characteristic *symptoms* are restlessness, crying, either continuously or in paroxysms, and the raising of the hand to the ear. In many cases there are convulsions of one or both sides of the body, opisthotonic flexion of the neck and back, squinting, and other signs of meningeal irritation. High temperature is usual, and Hartmann has drawn attention to the rapid loss of weight, which is soon recovered when the otitis is successfully treated. The examination of the tympanum presents almost insurmountable difficulty unless it is borne in mind that to expose the membrane the auricle is to be drawn downwards and not upwards. In cases of acute inflammation the swelling often merges so continuously into the wall of the short meatus that its outlines can scarcely be identified. Otherwise the description as given for the adult is applicable. In view of the importance of the diagnosis it is justifiable, in doubtful cases, to make an exploratory paracentesis, especially when "meningeal" symptoms are present.

The *treatment* is the same as in the adult. In the first instance, the instillation of carbolised glycerin is advisable, but paracentesis should not be delayed. The membrane is more resistant in the infant than in the adult, and, although drainage through the short Eustachian tube is relatively easier, dangerous extension may take place before the membrane yields, and paracentesis is, therefore, necessary on even slighter indications. This should be followed by gentle syringing with a solution of borax and boric acid and the instillation of drops of a saturated solution of boric acid. The nose and naso-pharynx should receive careful attention.

Acute Inflammation of the Attic.—The attic of the tympanum is sometimes the seat of a limited suppurative inflammation, having special characters depending on the anatomical peculiarities of the part; the most important being its close connexion with the antrum, its communication with the tympanum proper being in some cases extremely wide, in others very narrow, and the comparative isolation of that pouch known as Prussak's space or the external attic, which lies just above the short process of the malleus. This communicates with the rest of the attic by a narrow neck, generally behind, but sometimes in front. From the direct continuity of the attic with the antrum, suppuration in these two cavities often persists, even when the inflammation in the rest of the tympanum has subsided; indeed, localisation of the disease in the attic strongly suggests that there is active implication of the mastoid cavities, and forms an additional indication for opening them. This is all the more likely to be necessary if the communication between the epitympanum and tympanum is small and is still further narrowed by inflammatory swelling or adhesion. The neck of the external attic is sometimes obliterated by inflammatory adhesion, and the pouch continues as a suppurating cavity, which empties itself through a perforation in the membrane of Shrapnell into the external meatus. There appear to be cases in which suppuration in the external attic is excited by infection from the external auditory canal.

When the external attic is cut off by adhesions from the rest of the tympanic cavity, air driven up the Eustachian tube does not reach it, and no perforation-sound is obtainable. It is, therefore, a good rule to suspect suppuration in the external attic when there is a profuse purulent discharge and on perforation-sound. The interference with hearing may be comparatively slight, and therefore the presence of fairly good hearing would help to confirm the suspicion.

From what has been said, the *diagnosis* of acute inflammation of the attic will be easily understood. The pain and fever may be as marked as in any suppurative otitis, but the redness and bulging will be chiefly confined to the upper part of the membrane and the adjacent portion of the wall of the external meatus. The bulging may, however, be so considerable as to hang down like a polypus over the rest of the membrane and conceal it more or less entirely from view (Fig. 8, Plate XIII.). A moist exudation from its surface takes place, in which the swelling is bathed. On the occurrence of perforation the discharge becomes more profuse and there may or may not be a perforation-sound obtainable on inflation, according as the external attic or the epitympanum is shut off or not from the general tympanic cavity. When the perforation is limited to a small space immediately above the short process, the pus is probably confined to the external attic, but when it extends backwards to the tympanic ring, there is more probably a simultaneous implication of the antrum, more especially if the discharge be very profuse.

Prognosis.—Suppuration in the attic, if duly recognised and treated, will, in many cases, subside completely, but, as a rule, it is less amenable than suppuration in the general tympanum. It is apt to become chronic, and to present the serious aspects (cholesteatoma, caries, necrosis, etc.) described in the section on chronic suppuration. It may also lead to the various pyaemic and cerebral complications elsewhere described (p. 475). One of the less serious results is the formation of adhesions between the malleus and the outer wall of the attic, giving rise to fixation of the ossicle, with consequent impairment of hearing.

The *treatment* of acute attic suppuration differs only from that of general tympanic suppuration in the direction of the incision, which is made horizontally from before backwards through the swelling, and in the necessity of earlier resort to the mastoid operation. A pendulous protrusion may be removed by means of a snare or a miniature punch-forceps (Hartmann's). The various "drops," by preference alcoholic, may be coaxed into the attic perforation by suitable declination of the head, while the tragus is repeatedly compressed, and, during the stage of subsidence, they may be injected by means of Hartmann's or Milligan's intra-tympanic cannula (p. 459). If there is reason to suppose that the whole tympanic cavity is trying ineffectually to drain itself through the attic (auscultatory sounds indicating fluid in the tympanum) and the discharge persists, a counter-opening may be made through the tympanic membrane. The final drying is often hastened by the insufflation of powders, such as boric acid or iodoform, through the perforation.

Acute Suppuration in the Antrum and Mastoid Cells.—In every acute suppuration of the middle ear, the mucous membrane of the antrum and mastoid cells is simultaneously or consecutively affected, there being at first a simple empyema, and next, especially in infective cases or enfeebled individuals, implication of the bone of the nature of caries and, occasionally, necrosis. This is, of course, favoured by any obstruction to the escape of the discharge, and is most often the result of streptococcal invasion. The pneumatic spaces become filled with pus, and the dissepiments between the smaller cavities break down into a mass of granulation and soft bony tissue. The erosion may take place in an outward direction, especially in children, but often the outer walls offer considerable resistance, and the roof gives way in the direction of the dura mater of the middle fossa, the groove for the lateral sinus, or the digastric groove, leading to the typical extradural abscess, parasinuous abscess, and Bezold's mastoiditic abscess respectively. These forms of extension may be favoured by the occurrence of extreme thinness or actual dehiscence of the bone at the particular parts, as the result of peculiarities in the original formation.

Implication of the mastoid is usually evidenced by the pain in the region radiating over the side of the head, tenderness on pressure over the antrum just below the temporal line, and increase of the tenderness at the tip of the process, which is the rule in all acute middle-ear suppurations, but which should pass off in less than a week after free evacuation of the tympanum. Swelling of the tissues behind the auricle from infiltration usually follows, especially if the pus is working outwards. This increases to a bogginess, pitting on pressure, or even to a fluctuating swelling, with displacement of the auricle downwards and outwards when pus forms under the periosteum. The skin may give way with the formation of an external fistula. A swelling of the postero-superior wall of the external meatus is of the same significance, this being the anterior wall of the mastoid cavities.

If the bone gives way on the inner wall of the mastoid cells, the pus reaches the digastric groove and burrows down the side of the neck along the sheaths of the muscles, forming an abscess which may bulge in front or behind the sterno-mastoid, and may protrude into the pharynx. This is known as Bezold's mastoiditis (*vide* p. 466). It is apt to be mistaken for suppuration of the cervical lymphatics by those who are unfamiliar with its mode of origin, and it is sometimes accompanied by inflammation of these glands. Pressure on the swelling will often cause pus to well up from the middle ear or from the mastoid cavities if these are opened by operation, when also the opening into the digastric fossa may be detected by inspection or probing. Apart from such direct signs, implication of the mastoid cavities may be surmised, if the aural discharge persist without material diminution for more than four or five weeks, more especially if it be more purulent than mucous, or contains crumbs of bone, and if its quantity be greater than the tympanic cavity alone could secrete. Marked loss of strength, or the existence of any constitutional dyscrasia, especially diabetes, would heighten the suspicion. The blood-count often shews leucocytosis. The heightened specific gravity of the discharge, as tested by the benzole-chloroform method, has been stated by A. Forselles to indicate intramastoid ostitis. In very young children the pus finds its way out through the persistent squamo-mastoid suture, and even in older children it breaks through the outer cortex more readily than in adults.

Occasionally, especially in scarlet fever in children, instead of carious destruction of the bone resulting from acute suppurative otitis, a portion of bone undergoes necrosis, forms a sequestrum, and very slowly becomes loose. The affected portion is frequently the tympanic part of the temporal bone which forms the floor and walls of the meatus. Sometimes a large portion of the petro-mastoid segment is detached, laying bare the dura mater, whilst occasionally the labyrinth is more or less completely necrosed. The condition produces nearly the same local changes as are found in caries, but on incision or after the breaking-down of the soft parts the bare sequestrum is felt with the probe, and may be made out to be fixed or detached, but there is more pain caused by probing when the sequestrum is loose, as its angles are driven into the surrounding soft parts.

The *diagnosis* of caries or necrosis is fairly clear when the outward signs are present, either over the mastoid process or on the postero-superior wall of the meatus. Furuncle on the posterior wall of the meatus, which is often accompanied by slight infiltration and tenderness over the mastoid, may simulate this, but it can be distinguished by the lesser depth of the tender conical swelling, or swellings, in the meatus, by the absence of the characteristic signs of suppurative otitis media, by the preservation of hearing when the meatal swelling is gently compressed, by the increase of pain during mastication, and by the site of tenderness being below rather than behind the ear. Inflammation of a lymphatic gland in the mastoid region sometimes imitates genuine mastoiditis, but at first the gland is freely movable on the bone, whilst later it forms a fixed but shelving and circumscribed swelling, unlike the general infiltration of the soft tissues found in acute mastoiditis, or the extensive fluctuating swelling with downward and outward displacement of the auricle in periosteal mastoid abscess. Further, the cause of the adenitis is usually some obvious source of irritation of the scalp and not a suppuration of the middle ear.

Primary acute suppuration in the cortical mastoid cells, apparently without previous infection through the tympanum, is rare. It is characterised by throbbing pain in the mastoid region, local tenderness on pressure, and, after a few days, by infiltration of the superjacent soft tissues. It differs from the acute disease now under consideration only in the absence of evidence of implication of the tympanum. In all probability it is essentially the same disease, the cocci having been overcome in the tympanum but not in the mastoid, usually pneumatic, cells. When the cortical layer of the bone gives way, the pus may burrow just as in post-otitic cases. Acute primary mastoid periostitis is another affection occasionally met with, and presents similar symptoms, but tenderness and boggy swelling appear early, and the symptoms are at once relieved by incision down to the bone, which is not the case in otitic mastoiditis.

Mastoid swellings may be produced by gumma or by the outward extension of malignant disease when it has burst through the mastoid cortex. The diagnosis from malignant disease is very difficult, even when it is considered, as from its rarity it may not always be. The greater violence of the pain, the glandular infection, and the steady progress may suggest a diagnosis which is confirmed by the microscopical examination of portions of outgrowths into the meatus, or, as too often happens, by the appearance of the tissue when the mastoid swelling is incised on the supposition of its being inflammatory.

Mastoid neuralgia is sometimes a source of confusion. In it the pain is intermittent and is rather relieved than increased by pressure. It is attributed to the pinching of the nerves by chronic inflammatory sclerotic changes in the bone. The presence of neurotic or hysterical symptoms and the absence of fever or signs of otitis media help in the diagnosis. It is relieved by the removal of a superficial layer of bone in the mastoid region, so that the commencement of a mastoid operation, even in error, is sometimes the means of cure.

The diagnosis of mastoiditis secondary to acute from that secondary to chronic suppuration of the middle ear is of great importance, in view of the difference in the methods of treatment and operation required in each. Lermoyez points out the difficulties. Assistance may be obtained from the previous history and the appearances in the ear; but the patient's account of his past history is often fallacious, as he may have had chronic suppuration of the middle ear without any knowledge of its existence, and the changes in the ear, though often perfectly definite, as shewn by great destruction of the membrane or of bones, or by the presence of an unmistakable cholesteatoma, are frequently quite indefinite, and evidence of chronic suppuration may only be forthcoming when the cells have been opened in the belief that the otitis was acute.

The *treatment* of acute suppuration in the antrum and mastoid cells varies according to the degree and stage of the disease. If the signs only indicate the threatening of the extension of the suppuration to the bone, all that is necessary is to incise the membrane freely, or to enlarge the previous perforation, applying the cold coil, or, if it fail to relieve, hot compresses, and, if circumstances permit, washing out the tympanum through the Eustachian tube by a catheter. If, however, the local signs described above are definite, or if there are symptoms of impending or present cerebral or constitutional infection, the mastoid cavities must be opened by operation.

The following are among the chief symptoms of *threatening* intracranial or septicaemic complications:—Continued fever, with or without intermissions, in spite of free incision of the membrane (which should be made even if there is a perforation), and not accounted for by independent accompanying causes (for example, tonsillitis, naso-pharyngitis, pneumonia), calls for opening the mastoid in view of the imminence of infection of the lateral sinus, or septicaemia. Cerebral symptoms, such as delirium, restlessness, headache, vertigo, twitchings, with continuous high fever (in children, convulsions) arise from irritation or from threatening inflammation of the meninges, and if they persist in spite of free incision and are not with any probability due to pneumonia, enteric or other fever, or to tuberculous meningitis, the mastoid should be opened, in view mainly of the threatening of extradural abscess or meningitis.

Extreme giddiness, vomiting with subjective noises, and marked loss of hearing for the highest pitched tones, indicate impending extension to the labyrinth, and the necessity for complete opening of the mastoid and tympanic cavities by the radical mastoid operation.

The symptoms of actual extradural suppuration, leptomeningitis, sinus phlebitis, septicaemia, pyaemia, cerebral and cerebellar abscess as described elsewhere (p. 475), are urgent indications for the mastoid operation.

The continuance of the discharge without diminution, however gradual, after four or five weeks, is, as has been pointed out, a sign of implication of the bone, and, therefore, an indication for operation. (The operative treatment of mastoid disease is described on p. 469.)

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D. G.

CHRONIC SUPPURATIVE INFLAMMATION OF THE MIDDLE EAR

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SYN.—*Otitis media purulenta*; *Chronic and Pyogenic Otitis media*.

CHRONIC purulent disease of the middle ear is almost always the result of a past attack of acute suppuration. The various causes of acute pyogenetic otitis media are, therefore, the primary factors in the production of chronic disease. Certain local phenomena, such as the existence of an unduly small or a too highly situated perforation of the membrana tympani, tufts of exuberant granulation-tissue or intra-tympanic adhesions, by interfering with the mechanism of free drainage, are important factors in inducing chronicity. The exact moment at which an acute process passes into what is usually accepted as chronic disease is impossible to define accurately, and depends not so much upon the lapse of time as upon the existence of certain organic changes which have taken place in the mucosa of the tubo-tympanic tract.

The exanthemata, measles, scarlet fever, scarlatinal diphtheria, and small-pox; general diseases, such as influenza, pneumonia, diphtheria, mumps, pulmonary and laryngeal tuberculosis, enteric fever, malaria, Bright's disease, cerebrospinal meningitis, and various catarrhal affections of the upper respiratory tract, by causing acute suppuration of the middle ear are, therefore, the original factors in many cases of chronic disease. The extension of inflammation from the mucosa of the nasal passages, the nasal accessory sinuses, or the naso-pharynx very frequently causes acute suppurative disease of the tympanic mucosa, and hence indirectly chronic suppurative disease also. Post-nasal growths, more especially adenoid vegetations, by inducing recurring attacks of catarrhal inflammation, and interfering with the mechanism of the Eustachian tubes and preventing drainage, are factors of great importance in maintaining purulency. Certain external causes, such as injury, the entrance of sea-water into the auditory meatus, draughts, or the extension of an inflammatory process along the lining membrane of the external ear, by producing an acute otitis media, are also remote causes of chronic purulency. The too forcible use of Politzer's bag or the injudicious use of the nasal douche may, by rupturing the membrana tympani, also be the immediate cause of chronic disease.

Tuberculous disease of the middle ear is usually considered to run a chronic course from its commencement, its characteristic features being its painless and asthenic course, the presence of one or more perforations of the membrana tympani, and its tendency to the production of facial paralysis and enlarged periotic glands. With the exception of tuberculous lesions of the middle ear, it is probable that all other chronic cases have been preceded by an acute or subacute attack of inflammation; but in some cases of diabetic and influenzal origin the acute phase of the disease may be very short.

New-born infants sometimes suffer from a form of purulent inflammation of the middle ear, which is probably due either to changes in the embryonic tissue normally present in the cavity of the middle ear, or to the passage of liquor amnii up the Eustachian tubes during forcible attempts at respiration. Suppurative disease of the middle ear is more frequent in children and in young adults than in later life, mainly because the exanthemata and the various affections of the lymphoid structures—for example, diseased tonsils and adenoid vegetations—are more frequent at this period of life. Dentition, congenital syphilis, and catarrhal affections of the upper respiratory tract are also important causes of middle-ear disease in early life.

In chronic suppurative middle-ear disease the whole middle-ear cleft is affected, though from the formation of inflammatory adhesions there may be localised areas of suppuration.

Bacteriology of Chronic Suppurative Otitis Media.—The organisms met with in discharges from the middle ear are extremely numerous. In chronic cases the infection is invariably a mixed one. The predominating organisms are saprophytes. Numerous organisms with varying degrees of pathogenicity are met with, such as diplococci including Gram-negative cocci, namely, Weichselbaum's meningococcus, the gonococcus, and the *Micrococcus catarrhalis*, and also the Gram-positive pneumococcus. Streptococci and staphylococci are very frequently encountered, as also are certain putrefactive bacilli and the *Bacillus pyocyaneus*, *B. tenuis*. Tubercle bacilli, although occasionally present, are difficult to find in smears from the middle ear; they are most readily discovered in tufts of granulation-tissue removed from the fringe of advancing bone disease, or in sections of an enlarged periotic gland. The most virulent and the most destructive organisms are diplococci and streptococci. Staphylococci, whilst not by any means so virulent, are often exceedingly difficult to dislodge from the tissues, and are held by many to be one of the main factors in inducing a chronic condition.

Morbid Anatomy.—In the initial stages of an acute purulent otitis media there is marked dilatation of the tympanic capillaries followed by a considerable outpouring of serum and a fairly free leucocytosis. When the irritant is slight, desquamation results, but when of greater virulence, leucocytes are present in large numbers and act as phagocytes. As chronicity becomes established, the number of living leucocytes becomes markedly diminished, and the shed epithelium changes from a columnar to a flattened and much more squamous form. Instead of polymorphonuclear leucocytes, lymphocytes appear, indicating the presence of granulomatous changes, and ultimately, when the underlying cancellous bone is invaded, myelocytes are found in considerable numbers. As the result of increasing irritation, the chronically inflamed mucosa throws out buds of exuberant granulation-tissue wherewith to protect itself. These tufts are in many cases the forerunners of myxomatous granulation-polypi. Accompanying these various vascular phenomena, certain ulcerative changes result; they depend on the shedding of the epithelial elements of the mucosa, and on extension of the septic process to the deeper layers of the muco-periosteum, thus producing caries, or cario-necrosis, of the surrounding bony parietes. In this way various routes are opened up along which pathogenetic organisms may travel to more deeply-seated structures and organs.

Symptomatology.—The symptoms of chronic purulent middle-ear disease may be pain, impairment of hearing, tinnitus, vertigo, and disturbances of taste and smell. *Pain* is, as a rule, little complained of, except when intercurrent attacks of inflammation supervene, or when the exit of discharge is interfered with as the result of the presence of exuberant granulation-tissue, polypi, sequestra, stenosis of the meatus, or suppuration round a cholesteatoma. The advent of pain may, however, be the signal of the commencement of some severe and even fatal complication, and should therefore be regarded with great suspicion in genuinely chronic cases. When extension takes place towards the adjoining mastoid cells, pain is frequently localised to the area of the mastoid antrum. When more deeply-seated structures are invaded, the pain is, as a rule diffuse, and is of very little localising value.

Impairment of Hearing.—The degree of deafness met with in chronic cases varies within wide limits, and has very little relation to the size or situation of the existing perforation. Thus, in some cases with a very large perforation, if not almost complete destruction of the membrane, a high percentage of hearing power is retained, whilst in other cases with only a minute perforation the loss of hearing may be very great. The chief factors of importance in the maintenance of good hearing are the integrity of the stapedio-vestibular articulation and of the auditory nerve filaments, whilst the presence of secretion in the middle ear, intra-tympanic adhesions, an oedematous or polypoid mucosa, partial loss of the ossicular chain, and secondary implication of the labyrinth seriously affect the general powers of audition. Perforation of the membrana Shrapnelli is frequently unaccompanied by any defect of hearing power. *Tinnitus* is not, as a rule, a marked symptom in chronic purulent middle-ear disease. When it does occur, it is due either to a commencing stapedio-vestibular ankylosis, to the formation of intra-tympanic adhesions, to the pressure of pent-up secretions, to the presence of a cholesteatoma, or to secondary implication of the internal ear. *Vertigo* may be due to concomitant labyrinthine congestion, to pressure upon the footplate of the stapes or upon the fenestra rotunda, or to suppurative disease of the vestibule and semicircular canals. Although not a very frequent symptom, it is one of much importance and gravity. *Disturbances* of taste and smell, especially of the former, are frequent, and are due either to implication of the chorda tympani in its passage through the middle ear, or to the escape of putrid secretion along the Eustachian tube into the pharynx.

Objective Appearances.—The most striking feature of chronic suppurative middle-ear disease is the presence of a constant discharge. The amount and the characteristics of the discharge vary within wide limits. It may be thick, yellow, and purulent, or thin, watery, and almost white in appearance. It may be odourless or extremely fetid; sometimes also it is blood-stained, whilst occasionally it has a bluish tinge, due to the presence of the *Bacillus pyocyaneus*, or has a greenish colour, from the presence of the *B. fluorescens*. It may contain flakes of concentrically arranged masses of epithelium when a cholesteatoma happens to be present, or spicules of bone when some existing disease of the temporal bone is in progress. A careful cytological examination of the discharge is of practical value, as it sometimes indicates the character of the underlying morbid process, and to some extent the source of the suppuration (*vide* p. 374).

The appearance of the membrana tympani varies greatly. As a rule, there is one perforation only, although two, or even three, are seen in tuberculous cases. The perforation varies much in size, shape, and position; it may be so small as to be scarcely visible without the aid of a magnifying speculum, and, on the other hand, it may be so large as practically to occupy the whole membrane. It may be situated in the membrana Shrapnelli anteriorly, medially, or posteriorly. In the first position it is usually associated with chronic Eustachian obstruction, in the last with disease of the posterior end of the middle-ear cleft, and the medial position is commonly complicated with caries of the head of the malleus. In the membrana tensa the perforation may be anterior or posterior to the handle of the malleus, or may occupy the inferior segments of the membrane and be somewhat kidney-shaped. It may be situated centrally or peripherally, and may be circular, oval, reniform, or linear. The age of the perforation may be roughly gauged from its objective appearances; thus, in comparatively recent perforations the edges have a pinkish, somewhat vascular appearance, whereas in chronic cases they are thickened, indurated, and non-vascular, from the heaping up of epithelium. The more chronic the case, the thicker the edges of the perforation and the greater its tendency to assume a circular shape. In almost all cases the perforation is visible by objective examination. Sometimes the use of a magnifying ear-speculum assists the examiner. The appearance of a pulsating spot upon the membrana tympani practically indicates the presence of a perforation, but is much more frequently noticed in acute disease of the middle ear. Inflation, either by means of Valsalva's experiment, Politzerisation, or catheterisation, by driving air or secretion from the cavity of the middle ear, assists in the discovery of the position of the perforation. The smaller the perforation, the higher pitched the note.

Prognosis must be considered in its relation to the preservation of hearing power, to the cessation of discharge, and also in reference to the risk to life. The original cause of the suppuration, the general condition of the patient, the particular segments of the middle-ear cleft principally affected, and the duration of the disease are important factors, so far as the preservation of hearing power is concerned. Exanthematous otitis media is generally more destructive to the integrity of the ear than purely catarrhal otitis media, whilst, as a rule, the longer the disease has lasted, the greater the amount of organic change, and hence the greater the degree of deafness. Suppurative disease of the epitympanum is, on the other hand, frequently accompanied by very little loss of hearing power. The danger to life depends largely upon the presence or absence of disease of the surrounding bony parietes. In most chronic cases there is some implication of the underlying bone. The exact position of the caries, or cario-necrosis, is all-important; thus, disease of the roof of the middle ear, or mastoid antrum, is liable to be followed by an erosion extending into the middle or posterior fossa, disease of the posterior antral wall by suppurative phlebitis of the sigmoid sinus, and disease of the labyrinthine capsule by extension of infection to the base of the brain. Although, as a general rule, pathogenetic infection extends by way of an osseous erosion, it may take place quite independently of this, micro-organisms travelling along vascular or lymphatic streams to distant parts, where they produce embolic abscesses. The presence of chronic suppurative otitis media is, therefore, always a menace to life, more especially when accompanied by a lesion of the osseous framework. Purulent disease of the atrium of the middle ear is, as a rule, more amenable to treatment than disease of the epitympanum. The implication of the mastoid antrum and adjacent mastoid cells adds considerably to the difficulties and dangers of the disease.

Treatment.—The main requisites in the treatment of chronic suppurative middle-ear disease are the provision of free drainage, surgical cleanliness, and the application of the ordinary principles of antisepsis. In many cases the existing perforation will be found too small or too highly situated to drain the cavum tympani adequately. In such circumstances it must be freely enlarged, and as far as is possible maintained open in its whole length. Tufts of granulation-tissue blocking an existing perforation and mucous polypi demand removal by snare or forceps. The patency of the Eustachian tube should be maintained by careful inflation and removal of any nasal or post-nasal pathological condition likely to interfere with its due physiological function.

To cleanse the cavity of the middle ear and the contributory paths of infection, the following plan is of use:—(1) Inflation of the middle ear; (2) syringing with a suitable antiseptic lotion or with sterilised water; (3) suction by means of a Siegle's speculum or Sondermann apparatus; (4) inflation; (5) syringing or mopping with sterilised wool. Having in this way cleansed the middle ear as completely as possible, and having secured free drainage, one or other of the following plans of treatment may be adopted:—(1) The employment of fluid remedies, namely, antiseptic or astringent lotions; (2) the insufflation of finely pulverised antiseptic powders; (3) the aspiration of the contents of the middle ear by tampons of gauze.

Where the discharge is copious, and where the patient can only be examined at intervals, treatment by means of antiseptic lotions is preferable. To ensure the successful action of any particular antiseptic lotion it should be allowed to remain in the meatus (previously cleansed) for at least ten minutes twice daily. Solutions of glycerin of carbolic acid (5 per cent), resorcin (gr. vi ad ℥i), sulphocarbolate of zinc or sodium (gr. vi ad ℥i), nitrate of silver (gr. x ad ℥i), boracic acid (gr. xx ad ℥i), acetate of lead (gr. vi ad ℥i), perchloride of mercury (1 in 2000), peroxide of hydrogen (1 in 4) may with advantage be employed. If whilst the lotion is in situ the patient performs a negative Valsalva its entrance into the middle ear is facilitated. After the application of the lotion, the ear should be dried and packed with a strip of antiseptic gauze or a pledget of antiseptic wool.

Where the perforation is large, where the discharge is scanty, and when the patient is under observation, the insufflation of antiseptic powders, such as boracic acid, aristol, europhen, dermatol, iodol, iodoform, or iodoform and boracic (1 to 4) may be employed. Before their insufflation the meatus should be carefully cleansed and dried. Insufflations should be made once every two or three days, according to the amount of discharge present. They are contra-indicated if the perforation is small, if active inflammation is present, and in very young patients.

Aspiration by means of gauze tampons may be advantageously employed when the amount of discharge is not very profuse and when the patient can be kept under observation. A strip of plain sterilised, cyanide or iodoform, gauze is packed loosely into the meatus down to and in contact with the membrana tympani. This tampon is changed as frequently as it becomes soaked. The use of tampons is contra-indicated in acute exacerbations of the disease and in the presence of an otitis externa.

Complications of Chronic Suppurative Middle-ear Disease.—*Granulations.*—One of the most frequent effects of chronic suppurative otitis media is the formation of granulation-tissue within the cavity of the middle ear. These tufts of exuberant mucosa vary in size from that of the head of a pin to the size of a pea, and may originate from any portion of the tympanic mucosa. Histologically they consist of oedematous tissue interlaced with delicate blood-vessels, and covered, in the first instance at any rate, with a ciliated epithelium. They may or may not be associated with underlying caries of the temporal bone. Occasionally they spring from the dura mater, and project into the middle ear through a bony erosion of the tegmen tympani or antri. In course of time the action of gravity causes the granulation-tissue to become pear-shaped, and consequently pedunculated. Such pedunculated growths springing from the mucosa of the middle ear constitute ordinary aural polypi. The polypus most frequently met with is the ordinary mucous polypus, consisting of a delicate network of areolar tissue with numerous small round cells, thin-walled blood-vessels, and mucous glands (Fig. 4, Plate XIV.). The superficial covering of the growths, in its deeper portions at any rate, consists of columnar ciliated epithelium, but towards the surface, and where it is more exposed to atmospheric influences, of a stratified epithelium. As a rule one pedicle only is found, occasionally two, and in exceptional cases three are present. They spring from any portion of the tympanic mucosa; more usually, however, from the internal or posterior wall of the tympanum. At times they grow from the margins of a perforated membrane or from the mucosa lining the antrum, or even the mastoid cells. Fibrous polypi are comparatively uncommon. In structure they consist of a dense network of fibrous tissue interlaced with blood-vessels. They spring from the deeper layers of the tympanic muco-periosteum. Angiomatous polypi consist of a dense plexus of blood-vessels supported by a framework of connective tissue; they are very vascular, and bleed freely when removed. True myxomatous polypi are rare, and histologically consist of gelatinous mucous tissue.

Malignant polypi may be either carcinomatous or sarcomatous. Carcinomatous polypi occur as a rule in people past middle age, and are sometimes the outcome of long-continued suppuration. Rapid infiltration of the surrounding tissue is frequent, accompanied by excessive pain and enlargement of the periotic glands. Sloughing and spontaneous haemorrhage are common. Sarcomatous growths are occasionally met with in quite young children, even in infants. To all appearances they present at first like ordinary mucous polypi. Their rapid recurrence after removal and the liability to spontaneous haemorrhage should arouse suspicion as to their real nature.

The treatment of aural polypi consists in (1) removal of the growth: (2) destruction of its base or "root"; (3) treatment of the suppurative condition which in the great majority of cases is responsible for the growth.

(1) Removal of the growth may be affected by one or other or by a combination of the following methods: (*a*) syringing; (*b*) the use of lotions; (*c*) the application of caustics; (*d*) the use of snares, curettes, ring knives, or forceps. (*a*) Syringing.—In the case of polypi attached to the underlying mucosa by long thin pedicles, the act of syringing, especially if conducted with a certain degree of force, may be sufficient to dislodge the growth, either by the twisting and rupture of its pedicle, or as the result of subsequent sloughing. (*b*) Use of lotions.—Small, soft and oedematous granulation-polypi, if subjected for prolonged periods to the action of rectified spirit, occasionally shrivel up and disappear entirely. The ear is first syringed, then carefully dried with cotton-wool, after which it is filled with a warm solution of rectified spirit. This solution is to be retained in situ for from 15 to 20 minutes two or three times daily. The dehydrating effects of alcohol and its power of coagulating the albuminous elements of the growth render its application very serviceable in selected cases. If an undiluted solution of rectified spirit prove too irritating and too painful, it should be diluted to one-half of its normal strength, and gradually used stronger and stronger, until the pure spirit can be tolerated. Alcoholic solutions of formalin are also serviceable. (*c*) The application of caustics.—Various caustics are of value, namely, chromic acid, persulphate of iron, nitric acid, or trichloracetic acid. A crystal of chromic acid or of persulphate of iron is fused on to the end of a probe, which is passed down and pressed firmly against the growth. This leads to the formation of a slough of varying thickness. The slough is subsequently removed with a pair of forceps or syringed away, after which another application of the caustic is made, and so on, until the growth has entirely disappeared. In order to limit the action of the caustic, and to avoid injury to the cutaneous walls of the meatus, it is a useful plan to insert a small ring of thin india-rubber tubing into

the meatus and cauterise the growth through it. Instead of the application of crystals a cotton-tipped probe may be dipped in a solution of chromic acid (ʒij ad ℥i) or persulphate of iron (gr. xl ad ℥i) and applied as described above. Special care is necessary in the use of nitric acid on account of its great diffusibility. Trichloracetic acid has the disadvantage of causing a considerable amount of after pain. Before using any of the above-mentioned caustics, the meatus should be filled with a 10 per cent solution of cocaine or Gray's solution. Subsequently, to relieve pain insufflation of equal parts of orthoform and lycopodium powder is useful. Nitrate of silver, on account of its very feeble caustic action, is of little value. The efficiency of the alcoholic and caustic methods of treatment is much enhanced by a preliminary scarification of the growth. (*d*) Removal by means of instruments.—Small flat non-pedunculated growths are to be removed by means of sharp curettes, ring knives, or forceps. After preliminary cocainisation a ring knife, such as Politzer's, or a curette is introduced so as to encircle as much of the growth as is possible. By an upward movement of the instrument the growth is cut away close to its attachment to the underlying mucosa. It may be necessary to introduce the curette two or three times, so as to clear out the whole of the growth effectually, haemorrhage meanwhile being kept in check by pressure with pledgets of wool, syringing with hot sterilised water, or by instillation of cocaine and adrenalin drops. The root or base of the growth is now to be cauterised as previously described, until all trace of the growth has been entirely eradicated.

Polypi which have a long pedicle, and which fill the whole or the greater part of the external meatus, are best removed by the cold wire snare (Fig. 50). The loop of the snare having been previously opened out so as to encircle the growth readily, is passed into the meatus along its inferior wall, and is introduced well into the sinus of the meatus until definite resistance is encountered. It is then gradually tightened, an upward movement being executed at the same time, and is finally drawn outwards. In this way the growth is cut off obliquely and comes away in the loop of the snare, or is picked out of the meatus by means of a pair of angular forceps. The portion of the growth remaining within the cavity of the middle ear is ultimately destroyed by the application of caustics—for example, chromic acid and dehydrating lotions containing rectified spirit. The very careful application of a fine galvano-cautery point to the base of the growth is also a useful method of treatment.

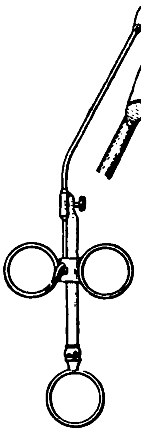

FIG. 50.—Cold wire snare for polypi.

After-treatment.—After the removal of the growth and arrest of haemorrhage, the meatus is to be packed with a strip of antiseptic gauze and a pad applied over the affected ear. Subsequent treatment is to be conducted upon the "dry" principle when possible,

PLATE XIV.

EUSTACHIAN OBSTRUCTION AND CHRONIC MIDDLE EAR CATARRH.

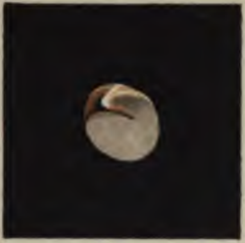

Fig. 1.
Indrawing of the membrane due to Eustachian obstruction (after McBride).

METHODS OF EXAMINATION AND GENERAL SEMEIOLOGY OF THE EAR.

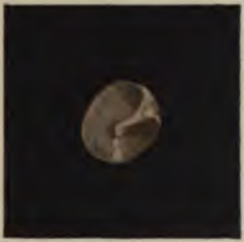

Fig. 2.
Right normal tympanic membrane.

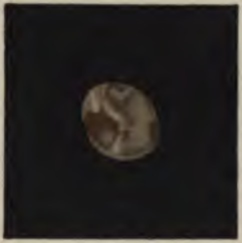

Fig. 3.
Left normal tympanic membrane of the more transparent variety.

CHRONIC SUPPURATIVE INFLAMMATION OF THE EAR.

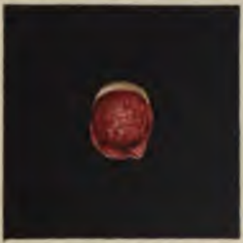

Fig. 4.—Polypus auris.

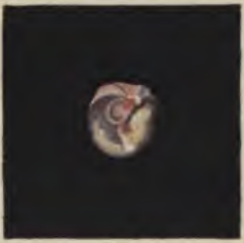

Fig. 5.
Calcareous deposit. Cicatrix.

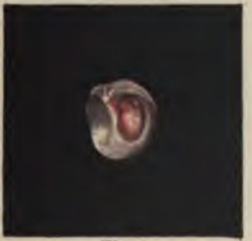

Fig. 6.
Perforation for artificial drum.

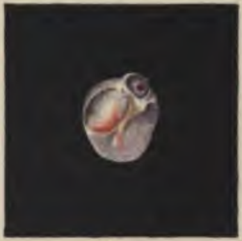

Fig. 7.
Perforation in Shrapnell's membrane.
Bare bone felt through perforation with probe.

the dressings being changed once or twice a week, according to the amount of discharge present. Should circumstances prevent such frequent dressings, drops containing rectified spirit are to be instilled once or twice daily, great care being taken to keep the parts as aseptic as possible. When all suppuration has ceased, efforts should be made to produce cicatrisation of the perforated membrana tympani.

Recurrence of Polypi.—The removal of polypi, if unaccompanied by careful after-treatment, actually favours their recurrence. If, however, thorough antiseptic treatment is instituted and kept up until all suppuration has ceased, recurrence is highly improbable provided that no disease of the underlying bone is present, and that the growth is benign. The repeated recurrence of aural polypi should therefore suggest the presence either of an underlying caries or necrosis of the temporal bone or of a malignant growth. Careful examination with a probe will usually confirm the presence of bone disease, whilst if a microscopic examination of the discharge reveals the existence of bone spicules or of numerous myelocytes the diagnosis can no longer be doubtful. The surgical treatment of malignant disease of the tympanic mucosa is eminently unsatisfactory, recurrence almost invariably taking place.

Caries and Necrosis.—A very common sequel of chronic suppurative middle-ear disease is caries or necrosis of the tympanic parietes or mastoid area; this is because the mucosa of the middle ear serves also as a muco-periosteum. Patches of caries may be quite minute or may extend over considerable areas of the surrounding bone. Necrosis is comparatively common in tuberculous and in gonococcal cases. Large portions of the temporal bone may become detached as sequestra, for example, the outer attic wall, the semicircular canals, the cochlea. In cases of suppuration of the recessus epitympanicus the outer attic wall, the head of the malleus, and the body of the incus are peculiarly prone to become affected and ultimately detached. The subjective indications of caries or necrosis are very indefinite. Pain of a deep-seated boring nature may be present. Vertigo and tinnitus are especially common when the outer labyrinthine wall is affected, and complete loss of hearing may result from a cochlear lesion. The main objective indications are the presence of a foul-smelling discharge hardly influenced by antiseptic treatment, and the presence of oedematous bone granulations. Palpation with a probe will usually reveal the existence of rough and softened bone, whilst a microscopic examination of the discharge may shew delicate spicules of bone and large numbers of myelocytes. Glandular affection is comparatively rare except in cases of tuberculous or malignant origin.

Epitympanic Suppuration.—One of the most troublesome forms of chronic middle-ear suppuration is disease of the recessus epitympanicus, or "attic" of the middle ear. This portion of the middle ear, about 6 mm. in height, is situated above the level of the short process of the malleus. Its outer wall is formed by the inner end of the superior osseous wall of the external meatus, and by the membrana Shrapnelli. It contains the head of the malleus, the body of the incus, and their ligamentous attachments, the two ossicles and their ligaments being so arranged as to divide the space into two compartments, a larger inner and a smaller outer. The outer attic is again divided into two spaces, an upper and a lower. The lower or Prussak's space is bounded above by the external ligament of the malleus and externally by the membrana Shrapnelli; whilst the upper, bounded above by the superior ligament of the malleus and below by its external ligament, is divided by reduplications of mucous membrane into a varying number of small compartments, any or all of which may become the site of suppurative disease. Suppuration in this region is peculiarly intractable on account of the difficulty in securing free drainage, and also on account of the liability to ossicular and parietal caries.

Epitympanic suppuration may be part of a generalised suppurative process affecting the middle-ear tract, and more especially its posterior or antral end; it may result from extension of inflammation of the external meatal walls, and frequently occurs in cases with long-continued Eustachian obstruction. Whatever be the exciting cause, and whether the suppurative process affects the inner or outer recesses, there is ultimately perforation of the membrana flaccida. Perforation in the anterior portion of the membrana flaccida is most usually associated with chronic Eustachian obstruction due to nasal or post-nasal causes. Central perforations are held by many to be the result of the extension of disease from the meatal walls, whilst perforations occupying the posterior portion of the membrane are generally associated with antral suppuration. In all cases the disease is apt to become chronic, and to be associated with ossicular or parietal caries and the formation of exuberant bone granulations (Fig. 7, Plate XIV.). The frequency with which the perforations are marginal favours the formation of an attic cholesteatoma as the result of ingrowth of epithelium from the cutaneous lining of the external meatus. The size of the perforation in such cases varies greatly; it may be little larger than the point of a pin, or it may occupy the greater portion of the flaccid membrane.

Symptoms.—Acute suppuration of the epitympanum is frequently ushered in by severe local and general symptoms. In young children the constitutional disturbance may be so great as to give rise to a diagnosis of meningitis. Imperfect drainage from the infected area is the main cause of chronicity in most cases.

In chronic cases there is as a rule no pain except when intercurrent attacks of acute sepsis ensue, or when the perforation becomes blocked by exuberant bone granulation-tissue with resulting retention of secretion. Tinnitus and vertigo are sometimes present, and are due either to increased intra-labyrinthine tension or to a secondary labyrinthine congestion. Acuity of hearing is often remarkable in cases of attic disease even in distinctly chronic cases. The objective indications of the disease comprise the existence of a perforation of the membrana flaccida, the frequent presence of exuberant buds of granulation-tissue, and ossicular or parietal caries.

The efficient *treatment* of epitympanic suppuration is hampered by the practical difficulties encountered in securing free drainage. Small perforations should therefore invariably be enlarged, and cleansing effected by means of specially constructed intra-tympanic syringes (Blake, Hartmann, Milligan) (Fig. 54). The injection of medicaments into the interior of the attic should be effected by means of specially constructed syringes (Blake) and under efficient illumination. Peroxide of hydrogen, boracic acid, or formalin is useful. In many cases the presence of caries of the head of the malleus, the body of the incus, or the outer attic wall, prevents the arrest of suppuration even after prolonged and careful after-treatment. Caries of the incus is particularly common in such cases.

Formerly considerable reliance was placed upon ossiculectomy as a means of arresting disease in these cases. The experience of most practical aural surgeons is that, although ossiculectomy affords temporary freedom from suppuration, it is seldom actually curative because the deeper portions of the middle-ear cleft, more especially the mastoid antrum, are implicated. In certain isolated cases, however, the operation may be advantageously performed.

The Operation of Ossiculectomy.—The patient is placed under a general anaesthetic, or has the membrana tympani well anaesthetised by Gray's solution, or by Neumann's method of subcutaneous injection of cocaine. An incision is made from the perforation right round the membrana tympani, which is in this way completely detached. The superior ligament of the malleus is now divided, and the tendon of the tensor tympani cut through. With an incus hook (Kretschmann, Ludewig) the incus is gently pulled downwards and outwards, and the incudo-stapedial articulation severed. The malleus and incus may now be removed by means of a delicate pair of forceps (Sexton) or with a snare. In many chronic attic cases the incus will be found to have disappeared; this is because it is suspended between the two other ossicles by means of delicate ligaments, and receives only one source of blood-supply from the tympanic blood-vessels. In such cases the malleus may readily be extracted by Delstanche's "Extracteur du marteau." The frequency with which the outer attic wall is carious in these cases renders it advisable to remove it at the same time. This may be accomplished either by means of a fine chisel or Pfau's forceps (Fig. 51). After excision of the ossicula auditus and removal of the outer attic wall all granulation-tissue should be scraped by curettes (Lake) and the mucosa swabbed with pure carbolic acid.

The stapes rarely becomes carious because it has a labyrinthine as well as a tympanic blood-supply. Its removal in certain circumstances has been recommended, and has been successfully carried out with apparently good results. It must be borne in mind, however, that there is always a risk in extracting the stapes during the progress of suppurative inflammation of the middle ear on account of possible implication of the labyrinth. In post-suppurative cases, however, in which the stapes has become more or less fixed by inflammatory adhesions its removal may result in an improvement in hearing power.

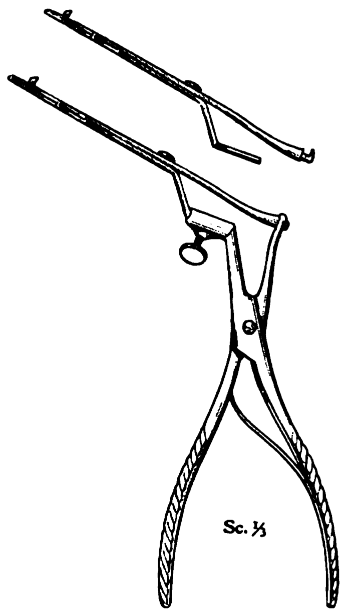

FIG. 51.—Pfau's forceps.

Post-Suppurative Sequels.—*Perforation.*—A not uncommon result of chronic suppurative otitis media is the formation of a dry perforation. In the majority of cases it is desirable to attempt to produce cicatrisation, partly on account of an improvement in audition which may thereby result, and partly on account of the lessened risks of recurrent suppuration which a healed membrane affords. In a recent case the edges of the perforation are fairly vascular and active, but in cases of long standing they are indurated and callous from overgrowth of epithelium from the dermic layer of the membrane, and until some means are adopted to remove this indurated epithelium cicatrisation is impossible. It must be borne in mind that in many cases improvement in hearing does not result even though healing takes place, and it is therefore necessary to differentiate cases in which it is advisable to attempt to produce cicatrisation from those cases in which at any rate the same necessity does not exist. This is ascertained by covering the perforation with a drop of glycerin or paroleine, or by carefully applying a paper or rubber disc over the opening; improvement in hearing indicates that an attempt to secure cicatrisation should be made, whereas the absence of any improvement makes it probable that there is some mechanical obstruction between the ossicula auditus, in the stapedio-vestibular articulation, or in the labyrinthine windows. In such cases it is very questionable whether it is worth while to make any attempt at cicatrisation.

Of the methods of treatment in vogue the most successful is the application of trichloracetic acid to the margin of the perforation (Okouneff). After a preliminary cocainisation or application of Gray's solution a cotton-tipped probe is dipped into a concentrated solution of the acid and applied with extreme care to the indurated edges of the perforation. The immediate effect is the production of a white eschar, which separates in a few days. If necessary other applications of the acid are made until a healthy edge to the perforation has been secured. As the result of the application temporary suppuration may be induced. The healing of the perforation takes place mainly by a concentric growth of epithelium from the dermic layer of the membrane, assisted to some extent also by the tympanic mucosa. The application of nitrate of silver, chromic acid, or pure carbolic acid is also serviceable. Radial incisions across the indurated margin of the perforation or complete removal of its thickened edge by the knife may also be tried. Large defects in the membrane may in this way be induced to heal. As the resulting cicatrix is always less resistant than the membrane itself, it assumes a saucer-shaped appearance due to the effects of the ordinary atmospheric pressure upon its surface, its edge being sharply defined and its surface highly refractive and slightly depressed (Fig. 5, Plate XIV.).

Where attempts to produce cicatrisation have failed, or where the membrane has been entirely destroyed, the employment of an artificial aid to hearing is frequently useful. Probably the best results are obtained by the use of Yearsley's cotton pads or Toynbee's artificial tympana. The cotton pad is made by rolling a piece of absorbent wool into the form of a cylinder or small ball. It is then moistened with carbolic oil, and carefully passed along the meatus until it impinges upon the head of the stapes or the stapedio-vestibular region. When first used such artificial drums should only be worn for an hour or so. As the ear becomes accustomed to their presence they may be worn during the day, but should invariably be removed at night. At times it is advisable to discontinue their use for a few days. Should they cause any recurrence of suppuration their employment should be temporarily suspended, and suitable astringent lotions should be prescribed. In some cases the healed or cicatrised portion of the membrane may become extremely lax and flaccid, with the result that not only is audition interfered with, but also the constant movement of the cicatrix is a source of annoyance to the patient. Incision of the cicatrix or puncture with a very fine galvano-cautery point may serve to tighten it up. The application of contractile collodion has also been employed (M'Keown).

Intra-tympanic Adhesions.—Chronic suppuration within the middle ear may set up adhesions between the membrana tympani and the inner tympanic wall, between the ossicula auditus, or between the footplate of the stapes and the margins of the fenestra ovalis. The general effect of such adhesions is to produce impaired mobility of the ossicular chain, and consequent interference with the passage of sound waves to the internal ear. In recent cases these adhesions may be successfully treated by forcible inflation of the middle ear, by pneumo-massage, or by means of Delstanche's raréfacteur, or by Sondermann's suction apparatus. More organised cicatricial tissue may in favourable cases be divided by means of suitably shaped knives, whilst in advanced cases the remains of the membrane and the ankylosed ossicular chain may be advantageously removed. Adhesion of the stapes to the margins of the fenestra ovalis—stapes ankylosis—at once the most important of intra-tympanic adhesions and the most difficult to deal with, may be treated by mobilisation of the stapes (Miot) or by its removal (Jack).

The instillation of solutions of bicarbonate of sodium, iodide of potassium, papain, pepsin, and so forth, is recommended by some as a means of softening or even digesting the intra-tympanic cicatricial tissue. Thiosinamin and fibrolysin have also been advocated for this purpose.

Areas of calcareous degeneration upon the membrane or upon the mucosa of the inner tympanic wall are common post-suppurative sequels. A frequent appearance is the presence of a patch of lime salts in front of and behind the handle of the malleus, and sometimes united so as to form a sort of miniature horse-shoe (Fig. 5, Plate XIV.).

Hyperostosis.—Long-continued suppuration from the cavity of the middle ear may result in the formation of bony growths either within the tympanum itself or upon the walls of the external auditory meatus. In the first instance a chronic periostitis is induced. This leads to the ultimate formation of new bone and to consequent stenosis of the meatus. Hyperostosis of the meatus most frequently occurs upon its posterior wall, and may attain such a size as to become a distinct danger by leading to retention of pus behind it (*vide* p. 401).

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SUPPURATION OF THE LABYRINTH

SYNONYM.—*Pyo-labyrinthitis.*

Pyogenetic infection of the labyrinth is due to one or other of the following causes:—(1) extension of pyogenetic disease from the cavity of the tympanum; (2) pyogenetic infection originating about the base of the brain and extending along the perivascular or perineural sheaths of the auditory or facial nerves; (3) deposition within the internal ear of pathogenetic organisms floating in the general blood stream; (4) injuries.

The most frequent, and from the practical point of view the most important pathway is by extension of infective disease through the inner tympanic wall. Organisms may gain an entrance into the internal ear either as the result of erosion of some portion of the osseous labyrinthine wall, or in consequence of maceration of the stapedio-vestibular articulation or the membrana secundaria covering the fenestra rotunda. The pathways of infection in order of relative frequency are the external semicircular canal, the fenestra ovalis, and the pars promontoria. In an analysis of 198 cases of labyrinthine suppuration, Victor Hinsberg found that in 61 the path of infection could be definitely established. In 27 of the cases a fistulous track was found in the horizontal semicircular canal; in 17 the infective organisms had reached the labyrinth by way of the fenestra ovalis, in 2 by way of the fenestra rotunda, in 3 through both fenestrae, in 7 through a fistula in the pars promontoria, and in 8 through a fistulous track in the posterior or superior semicircular canal.

Pyogenetic infection of the labyrinth is rarely the result of acute suppurative middle-ear disease. It is much more commonly found as the outcome of chronic suppuration of the middle-ear tract. Out of a total of 89 cases, collected by Hinsberg, 18 followed acute and 71 chronic suppurative otitis media. Tuberculous disease of the temporal bone or the presence of a cholesteatoma is a very frequent factor in the production of labyrinthine suppuration.

Injury is also by no means an uncommon cause of pyogenetic labyrinthitis. Fractures of the base of the skull, the introduction of foreign bodies into the ear, or their clumsy and unskilful extraction, and operative opening of the labyrinth, intentional or accidental, are sometimes followed by pyogenetic infection. An unskilful paracentesis of the membrana tympani for the relief of middle-ear suppuration has been known to produce suppurative labyrinthitis. The most common causes of injury to the labyrinth are the unskilful use of instruments during operative procedures upon the outer labyrinthine wall or during curetting of the tympanic mucosa, especially in the region of the fenestra ovalis. Since the head of the stapes is frequently buried in granulation-tissue it may during a curettement be easily dislocated with disastrous consequences.

Labyrinthine suppuration may be either diffuse or localised. Diffuse suppuration is prone to follow the entrance of pathogenetic organisms into the vestibule either by way of the fenestra ovalis or the fenestra rotunda. Localised suppuration is most frequently met with in cases in which the external semicircular canal has been slowly eroded as the result of the presence of a cholesteatoma. No hard and fast line can, however, be drawn, as diffuse labyrinthitis readily results from what may have long existed as a localised process. Localised suppuration is at times responsible for the formation of sequestra of various portions of the internal ear, for example the cochlea in part or in whole, or portions of one or more of the semicircular canals. In exceptional cases the whole internal ear has been exfoliated.

The **symptoms** of pyo-labyrinthitis vary according to the particular portion of the internal ear principally implicated, and according to the acuteness or chronicity of the infective process. Deep-seated boring pain referred in the first instance to the depths of the ear and ultimately radiating over the affected side of the head is usually complained of. This pain is increased by sudden movements of the head, loud noises, and to a certain extent by mastication or deglutition. In an acute case there is a pronounced rise of temperature accompanied by the ordinary febrile phenomena, such as rapid pulse and furred tongue.

In pyo-labyrinthitis there is a high degree of nerve deafness, except in those cases in which suppuration becomes localised in the region of the semicircular canal system. The upper tone limit is now especially implicated, whilst Weber's test will be found negative and Rinne's test positive. Tinnitus, although commonly present, is not as a rule an urgent symptom. Usually it is high-pitched, sharp and shrill, or distinctly musical. In chronic cases, especially in those of tuberculous origin, it may be entirely absent. Vertigo is as a rule extremely pronounced and frequently precedes deafness and tinnitus. It may be constant and may exist even when the patient is lying in bed, or it may be paroxysmal and readily aggravated by any undue exertion, stooping, or indeed by any sudden movement. It is invariably aggravated by turning the head towards the affected ear, by attempting to stand with the feet together, or by walking with the eyes shut. It is frequently accompanied by nausea, and in severe cases by persistent retching and sickness. (For static tests, *vide* p. 357.) Nystagmus is very common; it may, however, be extremely transitory, and is therefore probably frequently overlooked. As a rule, it is in a horizontal plane and is most easily detected when the patient looks towards the healthy side. In localised suppuration of the horizontal canal it appears only when the eyes are turned away from the healthy side; in diffuse suppuration of the labyrinth it takes place in all the positions the eyes may occupy. It is always accompanied by an inequality in the size of the pupils, the pupil upon the same side as the affected ear being dilated.

In chronic cases in which the progress of the disease is slow and insidious, and in which the destruction of tissue is very gradual, the symptoms detailed above, although present as a rule, exist in a much less characteristic form. Progressive loss of hearing in any case of chronic suppurative middle-ear disease, accompanied by a gradual contraction of the upper tone-limit and diminution of bone conduction, should direct attention to the probable implication of the internal ear.

Prognosis and Terminations.—The prognosis in all cases of labyrinthine suppuration is serious both as regards the risk to life and as to the loss of hearing power. Some acute cases are very rapidly fatal from diffuse purulent pia-arachnitis; other cases, on the contrary, especially those due to the exanthemata, recover slowly with ultimate complete and permanent deafness. In chronic cases the main danger to be anticipated is the gradual spread of pus towards the meninges or the cerebellum. So far as the function of hearing is concerned there is invariably cause for grave anxiety. The extent of deafness produced varies with the virulence of the original infection, the destruction of tissue, and the duration of the disease. Labyrinthine suppuration secondary to exanthematous middle-ear disease is more destructive than that due to ordinary pathogenetic infection, whilst cases of tuberculous origin, on account of the great destruction of bone which is prone to take place, are invariably very dangerous. Localised suppuration of the labyrinth is less dangerous than when the whole internal ear is affected.

Complications.—The most frequent complications are suppurative pia-arachnitis, abscess of the cerebellum, and thrombosis of the jugular vein due to the spread of pathogenetic organisms by the cochlear or vestibular venules. The paths along which infective organisms spread to the interior of the skull-cap are in order of relative frequency: (1) The perineural sheaths of the auditory or facial nerves; (2) the aqueductus vestibuli; (3) the superior or posterior semicircular canal; (4) Mouret's canal.

Treatment.—In subacute cases in which a sequestrum can be felt with a probe, and in which no urgent symptoms are present, regular antiseptic douching of the ear and the instillation of solutions of peroxide of hydrogen may be tried in the hope that the sequestrum may become sufficiently detached to permit of easy removal. Lotions of the various mineral acids in dilute solution are sometimes used in order to try to dissolve the diseased area of bone.

In the vast majority of cases, however, operative interference is necessary. The first essential is the performance of the radical mastoid operation with a free exposure of the outer labyrinthine wall. To effect this the facial spur must be freely cut away. The actual opening up of the labyrinthine cavity may be effected after the method laid down by Jansen, Hinsberg, Botey, or the author. In the Jansen operation the posterior branch of the external canal is located and opened. The canal is followed in an upward and forward direction into the vestibule. During the operation the greatest care must be taken to avoid injuring the Fallopian aqueduct, which in normal circumstances is situated 1½ mm. below the ampullary orifice of the external canal. Hinsberg, having completed a thoroughly radical mastoid operation, opens the fenestra ovalis and removes the stapes if it be still present. A small stylet is introduced into the vestibule to act as a guide, and the bony covering of the anterior limb of the external canal is cut away until the stylet is exposed to view. Botey opens the vestibule by taking as his landmark the anterior limb of the external canal, opening it, and enlarging the opening thus made anteriorly and superiorly. Should the stapes be still present he removes it; if not, he opens the fenestra ovalis and cuts away the posterior portion of the pars promontoria between the two fenestrae.

The author's operation or "bridge" operation is as follows:—A complete and radical mastoid operation is first performed, and in order to secure free access to the whole area of the operation the following method of making a flap from the soft parts is adopted. A long knife is introduced into the meatus and made to cut vertically outwards along the line of junction between the superior and posterior cartilaginous walls of the meatus, the incision being carried well into the concha. The knife is now swept round in a circular direction parallel to the curve of the antihelix to the floor of the meatus. The comparatively large flap thus secured is trimmed and folded downwards and backwards on to the floor of the excavated mastoid process. The flap is kept in position by fine silk-worm gut strands passed first through the skin of the neck, then through the flap, and back again to a point close to the original point of entry, and tied over a rubber tube. The petro-mastoid is now entered in the triangular space between the posterior limb of the external canal and descending limb of the posterior canal (Fig. 52). This little triangle is situated 4 mm. behind the highest point of the extreme convexity of the descending portion of the Fallopian aqueduct. The opening thus made is enlarged upwards and backwards until the posterior canal is freely opened. By now working forwards along the external canal its anterior limb is opened, and by following this the vestibule is ultimately reached. A specially designed "facial protector" (Fig. 53) is placed in situ and made to lie over the aqueduct in the form of a cap. The fenestra rotunda is now located, opened by means of a small burr, and the pars promontoria cut away in an upward direction until the fenestra ovalis is reached, the stapes being removed if still present. By means of a specially constructed burr, cutting vertically, the bone lying immediately under the aqueduct is cut away so that finally, when the facial protector is removed, the aqueduct appears like the arch of a bridge facing the operator,

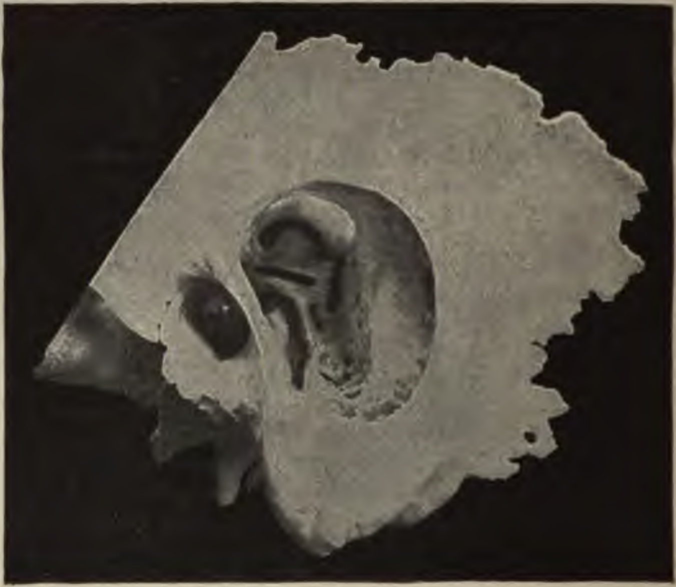

FIG. 52.—Photograph shewing the field of the author's "bridge" operation.

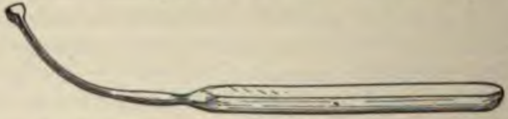

FIG. 53.—The author's "protector" for the facial nerve.

end on as it were, between the semicircular canal system behind and the cochlear system in front. Very free drainage of the various segments of the internal ear is thus effected, whilst the cavity is allowed to granulate and finally to obliterate itself. After the completion of the operation, and when the auricle has been put back into position (not necessarily sewn back), the edges of the incised concha are trimmed in such a way that when the finger is placed in the enlarged meatus its edge is found to be on the same level as the floor of the excavated mastoid.

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Tuberculous Otitis Media.—Suppurative disease of the middle-ear in tuberculous subjects is not necessarily of tuberculous origin. From their lowered vitality and the peculiarly sensitive condition of the mucosa of their upper respiratory tract such patients are particularly prone to attacks of suppurative middle-ear disease. On the other hand a tuberculous inflammation of the tympanic mucosa is by no means uncommon amongst those suffering from pulmonary or other forms of tuberculous disease. Since the discovery of the tubercle bacillus by Koch in 1882 the existence of tuberculous otitis media has been placed upon a definite and scientific footing.

Morbid Anatomy.—In a few cases small isolated yellowish-white tubercles deposited upon the tympanic mucosa have been visible to the naked eye; these nodules rapidly break down, and form minute ulcers which are apt to coalesce and to spread deeply. The amount of destruction of tissue is difficult, if not impossible, to gauge accurately by objective examination alone. In some cases the membrana tympani is almost entirely destroyed, in others it shews two or more perforations, and in exceptional cases it presents a sieve-like appearance due to the original presence and subsequent breaking down of numerous minute tubercles. The tympanic mucosa may be so ulcerated as to expose considerable areas of the underlying bone, and the ossicular chain may be partially or completely destroyed. In severe cases the cancellous tissue of the petro-mastoid may be entirely destroyed, and its place taken by oedematous granulation-tissue and purulent debris. The Fallopian aqueduct is prone to become opened up by carious erosion, and hence facial paralysis or paresis is often an early symptom of tuberculous otitis media. Sequestra consisting of portions of the cochlea or semicircular canals are by no means uncommon, whilst destruction of the labyrinthine windows and consequent infection of the internal ear are met with in from 20 to 30 per cent of the cases. In the later stages of the disease severe and fatal haemorrhage may result from exposure and subsequent ulceration of the internal carotid artery, jugular bulb, or lateral sinus. The dura mater in the immediate neighbourhood of the tuberculous focus becomes thickened, ulcerated, or studded by numerous oedematous granulations. At a very early period of the disease the periotic glands become infected and enlarged. Large masses of glands may thus form around the infected ear and become matted together by repeated attacks of periadenitis. Ultimately these glandular masses break down to form unhealthy abscess cavities, and a general systemic infection may result.

Etiology.—Tuberculous otitis media is most frequently found amongst patients suffering from pulmonary phthisis, and more especially during the later stages of the disease, when the resisting power of the tissues is at its lowest. In some instances it occurs in patients with tuberculous disease of joints, glands, tonsils, and naso-pharyngeal adenoids. There is considerable difference of opinion on the question whether tuberculous disease of the middle ear is ever primary. It is, however, becoming gradually accepted that primary tuberculous otitis media is not very uncommon, and the observations of Haug, Knapp, MacCuen Smith, the author, and others accentuate this point. Implication of the middle-ear tract is usually the result of aerial infection, the bacillus being carried per tubam. In certain other cases the infection is probably haematogenous, the bacillus being deposited within the tissues by the blood stream. This is especially prone to happen in tuberculous disease of bone and in miliary tuberculosis. The main disposing causes are residence in unhealthy, ill-ventilated, damp, and sunless rooms, insufficient nourishment, and a lowered vitality consequent upon exhausting diseases, such as scarlet fever, enteric fever, measles, diphtheria, and scarlatinal diphtheria.

Symptoms—Acute tuberculous disease of the middle ear is ushered in by symptoms almost similar to those of acute septic infection, the main differences being that the pain is not so severe, discharge is not so copious, and as a rule there is more than one perforation. In the more usual type of case the morbid process is asthenic from the commencement. Pain is as a rule absent, the first sign of the disease being the discovery of a thin, fetid, and sometimes sanious discharge from the middle ear. There is also usually considerable impairment of hearing, due partly to early and extensive disintegration of the contents of the middle ear, and partly to a concomitant infection of the labyrinth. Tinnitus is common, whilst vertigo is somewhat exceptional. At an early stage there is marked loss of taste upon the corresponding side of the tongue. The principal objective indications are the presence of one or more perforations with grey, unhealthy, and inactive edges. These perforations are prone to coalesce, with the result that a large defect in the membrana tympani is produced. A very frequent situation of such a tuberculous perforation is in the posterior superior segment of the membrane.

Diagnosis.—Certain clinical signs and symptoms, such as the painless appearance of a discharge from the middle ear, the presence of one or more perforations of the membrana tympani with indolent and inactive edges, the early onset of facial paralysis, and enlargement of the periotic glands, suggest the tuberculous nature of the affection. To prove this a careful search should be made for the tubercle bacillus either in pus taken from the middle ear, in tufts of granulation-tissue from the advancing edge of the disease, or in sections made from the enlarged periotic glands. The bacillus is, however, unquestionably difficult to find; and further, is readily mistaken for other acid-fast bacilli, such as the smegma bacillus, butter bacillus. Consequently, inoculation experiments may be necessary to establish the exact nature of the disease.

Prognosis.—Generally speaking, the prognosis is unfavourable, more especially in acute cases, in cases accompanied by early facial paralysis, enlargement of the periotic glands, and labyrinthine invasion. Its onset in advanced cases of pulmonary and other forms of tuberculous disease renders the outlook very grave. In primary disease of the antro-tympanic mucosa, or of the osseous tissue of the pars mastoidea, the prognosis, although grave, is by no means hopeless. In infants and very young children the outlook is distinctly bad. Intracranial lesions, such as meningitis, thrombosis and abscess, intestinal tuberculosis, and general marasmus are complications frequently encountered in such cases.

Treatment.—When secondary to advanced tuberculous disease of other organs little can be done beyond maintaining free drainage from the infected area and the employment of antiseptic treatment. Open-air treatment should be strongly insisted upon, especially residence in a dry, bracing, and sunny atmosphere. A nutritious diet, plenty of milk food, and suitable tonics should be prescribed. Any operative interference beyond the establishment of free retro-auricular drainage is to be deprecated.

In tuberculous disease of the middle ear and its accessory cavities, either primary or secondary, in young and debilitated children, the removal of areas of infected bone and the rigid employment of antiseptic treatment are frequently followed by recovery. In such cases no formal operation is advisable, the surgeon aiming in the first instance at the removal of carious or necrotic bone and the provision of free drainage. On account of the extremely debilitated condition of many of the patients it is advisable to operate in "stages." Where the chances of preserving any serviceable amount of hearing power are slight it is better to disregard the function of audition entirely and to endeavour to secure the filling up of the antro-tympanic cleft with healthy granulation-tissue. The removal of the enlarged periotic glands is very troublesome and tedious, and is much facilitated by a preliminary ligature of the internal jugular vein.

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Cholesteatoma.—The *pathogenesis* of cholesteatoma has given rise to considerable divergence of opinion. That it may occur as a primary formation, a fetal inclusion within the cavity of the middle ear or its accessory sinuses, is undoubted. Lucae and Kuhn have described cases in which the primary origin of a cholesteatoma would appear to be beyond question. Much more frequently, however, cholesteatoma is the outcome of previous suppurative disease of the tympanic or antral mucosa. Several of the older pathologists, notably Virchow, regarded cholesteatoma as a heteroplastic tumour, von Tröltsch considered it to be a retention tumour, Wendt as due to a desquamative inflammation of the tympanic mucosa, whilst Habermann and Bezold referred its formation to the inward growth of the epithelial lining of the external auditory meatus through a perforated membrana tympani. A combination of the two last hypotheses probably explains its mode of origin. Excessive growth of the epithelium lining the meatus in association with a marginal perforation and some obstructive lesion within the middle or external ear hindering the outflow of the products of a purulent inflammation are the factors principally responsible for its formation. An obstructed Eustachian tube, by leading to a lowered state of vitality of the tympanic mucosa, favours the ingrowth of the epithelium lining the meatus. Cholesteatomatous formations are also sometimes due to the ingrowth of epithelium through a fistula in the mastoid cortex or posterior meatal wall (secondary cholesteatoma). Politzer has, however, shewn that such cholesteatomatous masses may arise within the middle ear without the aid of any ingrowth of epithelium from the meatus; his reasons for this opinion are, first, that many of the cells composing the cholesteatomatous mass are non-nucleated, whilst the dermic cells of the external auditory meatus are nucleated, and secondly, because in many cases of exhausted suppuration within the cavity of the middle ear he has demonstrated microscopically an inward growth of epithelium without any cholesteatomatous formation.

Morbid Anatomy. — The masses consist of concentrically arranged layers of stratified epithelium, nucleated and non-nucleated cells, fat granules, cholesterin crystals, cocci, and purulent debris. In size they vary enormously. Within the substance of the tympanic mucosa they may be no larger than the head of a pin, whilst within the mastoid area they may attain the size of a walnut, and, by continuous pressure, open up the cavity of the labyrinth, the middle or posterior fossa, or penetrate the mastoid cortex and appear under cover of a subperiosteal mastoid abscess. In some cases of cholesteatomatous formation caries is present, in others it is absent; it may precede the formation of the mass or may be subsequently induced by suppuration around it. Extensive excavation of the temporal bone, sometimes followed by spontaneous extrusion of the cholesteatomatous mass, is by no means uncommon. Thus, as the result of the absorption of the posterior wall of the bony meatus, the antro-tympanic cavity and the external auditory meatus may be thrown into one; or the petrous bone may be hollowed out with complete destruction of its acoustic or static segments. In the early stages of cholesteatomatous growth the form of the mass approximates to that of the cavity in which it originates. At a later stage, as the result of gradual enlargement, pressure, and erosion, the bone becomes excavated, a more or less circular cavity with smooth walls and lined by a delicate layer of highly refractive squamous epithelium being formed.

Symptoms.—Cholesteatomatous masses may lie dormant within the epitympanum or mastoid antrum for varying periods without causing any distinctive symptoms. Their presence is, however, always associated with an element of danger; at any time suppuration may take place around the mass of decomposed epithelium and purulent debris, and lead to severe pain, high temperature, and symptoms referable to the particular portion of the temporal bone and surrounding soft parts which has been invaded. Spontaneous extrusion of a cholesteatoma may occur, especially when the mass has formed within the epitympanum and in association with a large perforation of Shrapnell's membrane. In rare cases they have been known to pass along the Eustachian tube and appear in the naso-pharynx. When in the mastoid antrum they may gradually erode the mastoid cortex and present externally within a subperiosteal abscess which is simultaneously produced. In other cases they perforate the posterior meatal wall through a previously existing fistula. In such cases symptoms referable to the particular route taken by the mass in its efforts of self-extrusion will be manifest. In many cases the cholesteatomatous mass erodes the tegmen antri et tympani, comes to lie in direct contact with the basal dura, sometimes perforates it and even invades the surrounding cerebral substance. Even the dense external labyrinthine wall is unable to resist its advance, and cases of erosion of the external semicircular canal and posterior portion of the vestibule are by no means unknown (Jansen, Milligan). Fatal results follow the presence of cholesteatomas either from general sepsis or from such intracranial complications as meningitis, brain abscess, infective thrombosis, or diffuse suppurative encephalitis induced by a preceding or accompanying caries of the surrounding bone, or by perforation of the mass into the interior of the skull or labyrinth.

Diagnosis.—The only certain diagnosis of cholesteatoma, before operation, is the discovery by inspection of a portion of the mass protruding through a perforated membrane or fistula in the posterior meatal wall, or the finding of acid-fast epithelial squames, arranged more or less concentrically, in the washings from the middle ear. The repeated appearance of such acid-fast squames after careful cleansing is important, in that it indicates the breaking down of some deep-seated mass. The presence of a cholesteatoma situated deeply within the mastoid area can only be definitely proved during the course of an operation, although its presence may have been suspected on account of protracted suppuration from the middle ear attended by imperfect drainage, and evidences of marked epithelial proliferation of the cutaneous lining of the osseous meatus. The masses of epithelium which are extruded spontaneously, or are washed out from the ear, appear as small pearly-white flakes, and have the microscopic structure already mentioned on p. 457.

Prognosis.—The presence of a cholesteatoma is always attended with danger to the patient. The cure of suppurative disease of the middle ear is impossible until after its removal, and its tendency to erode and to penetrate surrounding structures adds an element of danger which must not be forgotten. The more deeply it is situated and the more frequently suppuration develops around it the greater the risks. Spontaneous cure is so exceedingly rare that for practical purposes it need not be considered. Even after operation cure may be difficult to obtain, because extensive proliferation of epithelial cells may have taken place into the Haversian canals and canaliculi of the surrounding bone.

Treatment.—In cases in which the perforation of the membrane is small and situated towards its upper pole it is advisable to enlarge it by means of a free incision. Forcible syringing with an ordinary ear-syringe may then succeed in detaching the mass and cleansing the middle ear. Should the mass adhere closely to surrounding tissues it is possible by means of small curettes, scoops, or probes to break it up, after which syringing will be more effective. A useful instrument for this purpose, especially when the mass lies within the epitympanum, is Lake's curette. By attaching a small rubber tube to the end of the syringe and introducing it within the cavity of the middle ear a more direct stream of fluid is made to bear upon the affected parts. Preliminary instillations of rectified spirit or of salicylic acid in aniline oil facilitate the extrusion of the mass. The employment of such syringes as Blake's, Hartmann's, or the author's (Fig. 54) is indicated when the mass is situated within the epitympanum or Prussak's space. With the author's syringe a continuous flow of fluid under pressure is obtained, and the effects produced can be watched by direct inspection. The point of the syringe is introduced through the perforated membrane and is made to impinge directly against the cholesteatomatous mass. The pressure of the fluid is regulated according to the height of the reservoir. Syringing is kept up until the returning fluid comes away free from debris. This method of syringing has the further advantage of being practically painless.

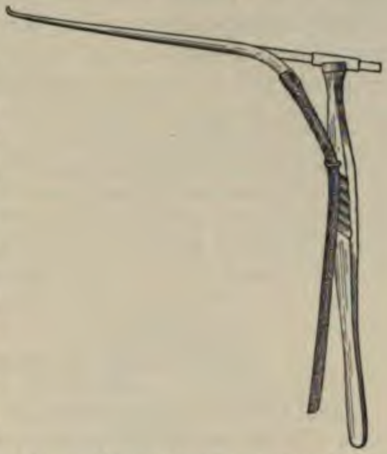

FIG. 54.—Milligan's intratympanic syringe.

In certain cases the expulsion of cholesteatomatous masses is facilitated by irrigation of the middle ear through the Eustachian tube. Solutions of bicarbonate of sodium, peroxide of hydrogen, or of normal salt solution are driven along an ordinary Eustachian catheter into the cavity of the middle ear. Portions of the mass are thus forced out by the *vis a tergo*, and other portions by spontaneous extrusion due to swelling from imbibed moisture. By such means it is possible to remove small masses when favourably situated. Their recurrence is, however, almost inevitable, owing to the existence of an underlying suppurative condition within the middle ear. Regular instillations of alcoholic solutions of boracic acid, salicylic acid, rectified spirit, or solutions of hydrogen peroxide, are useful in helping to arrest both the suppurative and desquamative processes which are present.

Where the cholesteatomatous mass is situated within the mastoid antrum or surrounding mastoid cells syringing is practically valueless, and operative measures must be employed.

As has been previously stated, the existence of such masses within the accessory cavities of the middle ear is fraught with danger on account of the risks of sepsis and of intracranial complications. In certain cases the removal of the external attic wall and the opening up of the antrum may suffice, in other cases it is in addition necessary to ablate the surrounding mastoid cells freely.

On account of the tendency to recurrence Reinhardt has proposed the maintenance of a permanent retro-auricular fistula through which the cavity within the mastoid process may readily be inspected and, if necessary, further treatment carried out. Of late it has been suggested (Dundas Grant, A. Hartmann) to leave the "matrix" of the cholesteatomatous mass to form the permanent lining of the cavity exposed by operation. As the "matrix" is, however, the product of a diseased condition, and as histological observation has shewn that epithelial processes dip down even into the Haversian canals of the bone, the suggestion appears to be fraught with danger. To minimise the liability to recurrence every visible shred of epithelium should be removed and the cavity subsequently grafted or allowed to fill up with healthy granulations.

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Diseases of the Mastoid Process

Acute Primary Periostitis of the Mastoid Process is distinctly rare. It occurs more frequently in adults than in children, and is due to exposure to cold, blows upon the part, or forcible pulling of the ear. Sometimes no obvious cause is found. It may remain as a localised inflammation, or may spread over the whole side of the head, producing an amount of oedema sufficient to cause closure of the eyelids upon the affected side. Should it extend downwards towards the apex of the mastoid process it involves the corresponding sterno-cleido-mastoid muscle, thereby interfering with the movements of the head. Suppuration may or may not result.

The symptoms are pain, at first local, but very shortly spreading over the side of the head, increased by pressure and by movements of the head. No impairment of hearing is as a rule observed except in cases with some intercurrent affection of the middle ear, or in which the stenosis of the meatus induced by prolapse of the postero-superior meatal wall is so great as to offer a mechanical obstruction to the entrance of sound waves to the membrana tympani. The patient's temperature is slightly elevated, more so if suppuration is taking place, whilst the surface temperature of the affected area is always raised.

Signs.—In all cases there is considerable oedema of the soft tissues over the mastoid, and marked pitting on pressure. When suppuration ensues the swelling is increased and the overlying skin presents a dull livid appearance. The external ear is thrown downwards, forwards, and outwards, and the amount of oedema is sometimes so great as to close the external auditory meatus. In exceptional cases congestion and slight swelling of the membrana tympani are noted.

Course and Termination.—In favourable cases oedema subsides and resolution takes place within three or four days. In other cases an abscess forms between the periosteum and the bone, and, unless opened, discharges superficially or into the external auditory meatus through one of the fissures of Santorini. Secondary necrosis of the mastoid cortex, with subsequent exfoliation of a thin plate of bone, has been observed (Politzer).

Diagnosis and Prognosis.—A diagnosis is as a rule easily made by a consideration of the local appearances and by the absence of deafness and any objective indications of middle-ear disease. In those cases in which the inflammatory process runs a practically chronic course the possibility of intramastoid suppuration must be borne in mind even in the absence of any objective indications of middle-ear disease. The prognosis is good except in delicate and "strumous" individuals.

Treatment.—In the early stages of the disease the local application of cold by means of a specially shaped ice-bag or a Leiter's coil (Fig. 55) is useful. Painting the swelling with tincture of iodine or a 5 per cent alcoholic solution of ichthyol, or gentle inunction with a 5 per cent oleate of mercury ointment, is at times successful. When there is marked oedema, uninfluenced by the application of any of the previously named remedies, a Wilde's incision is called for. When suppuration has taken place an incision from 1 to 1¼ inch long, parallel to the attachment of the auricle and ¼ inch behind it, is made down to the bone throughout its entire length, and the subperiosteal cavity cleansed and packed with gauze. A few stitches are introduced and tied quite loosely. When all suppuration has ceased and all oedema has disappeared the edges of the wound are brought into accurate apposition and the stitches tightened.

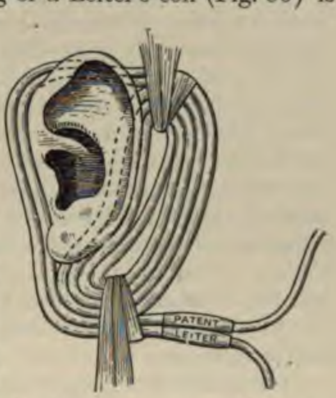

FIG. 55.—Leiter's coil applied to the ear.

When discharge has taken place from the meatus it may be necessary to enlarge the fistulous opening in order to secure a free exit for pus and to facilitate the introduction of a gauze dressing. The application of an elastic bandage over the ordinary dressing is useful in maintaining a close approximation of the superficial soft structures to the underlying bone.

Acute Primary Inflammation of the Mastoid Cells, although rare, sometimes appears after an acute middle-ear suppuration has run its course. It may also be due to injury, to cold, or to secondary infection from the naso-pharynx. It occurs most frequently in pneumatic processes, and generally attacks cells situated posteriorly and inferiorly.

Symptoms.—There is considerable local pain at first without any external evidence of swelling or oedema. The nearer the surface the inflammatory process is situated the earlier the external indications of its presence. Should the inflammatory process proceed to suppuration the pain becomes more severe, is greatly increased on pressure, and is accompanied by marked fever. External indications of importance are swelling and oedema of the superimposed soft tissues accompanied by displacement of the auricle. In ordinary circumstances the abscess breaks through the cortical layer, producing a subperiosteal collection of pus, but occasionally it breaks through the fissura mastoidea squamosa. In cachectic or syphilitic patients a definite cario-necrosis is frequent.

Prognosis is as a rule quite good except in debilitated patients or those suffering from a severe syphilitic dyscrasia.

Treatment.—Early antiphlogistic treatment, the application of ice, or the performance of a Wilde's incision is called for. When a definite subcortical collection of pus has taken place the infected area should at once be opened up and drained.

Acute Purulent Inflammation of the Mastoid Cells.—In every case of acute purulent inflammation of the tympanic mucosa pus will be found in the cavity of the mastoid antrum and in the adjacent pneumatic spaces. This is partly due to continuity of tissue and partly because when the patient is recumbent pus flows backwards towards the mastoid area. The antrum may in these circumstances act merely as a reservoir for the accumulating secretion, and provided that no retention or reinfection takes place, the case may go on to complete recovery without any mastoid symptoms whatever. On the other hand, should retention of secretion be brought about by swelling of the mucosa of the "iter ad antrum" or of the lining of the pneumatic cells communicating with the antrum, or should the mucosa or bony trabeculae of the cellular system of the mastoid process become definitely infected, an acute mastoid empyema results.

Pneumatic processes are much more frequently affected than diploic, whilst in certain diseases such as influenza, tuberculosis, scarlatinal diphtheria, diabetes, there is a much greater risk of infection than in ordinary pathogenetic infection of the middle-ear tract. Inefficient drainage of pus from the cavity of the middle-ear, due either to the presence of a minute perforation of the membrane or to the formation of granulation-tissue, is also a common cause of suppurative endomastoiditis.

Symptoms.—The earliest indication of the presence of acute inflammation of the accessory spaces of the middle ear is pain, at first local but rapidly radiating over the corresponding side of the head. This pain, which is of a deep-seated boring nature, is almost always increased by percussion and invariably by localised pressure over the antral area, the tip and anterior wall of the process. At the same time there is a rise of temperature, especially noticeable in children, in whom it may reach 103° to 104° F., rapid pulse, and other febrile phenomena. This acute inflammatory attack may resolve or it may end in the formation of an acute abscess. Resolution is more likely to take place in simple pathogenetic infections of the middle-ear tract, an acute abscess when the ear trouble occurs in the course of influenza, measles, scarlet fever, and similar diseases. In the initial stages of abscess formation there may be several small purulent foci, separated from one another by areas of healthy bone tissue. As the disease progresses these foci increase in size, and by becoming confluent, form a single large abscess cavity. The formation of an endomastoid abscess may be clinically recognised by an increase in pain which is as a rule persistent and localised. It is worthy of note, however, that the actual location of pain does not always correspond with the site of the abscess cavity. In the early stages of the disease the body temperature is elevated but does not necessarily remain so, even in cases in which the abscess is attaining a large size. In fact, not uncommonly the disease progresses while the temperature remains practically normal. The local temperature of the affected area, however, is always elevated. When the abscess formation has taken place subcortically there is at an early stage external evidence of its presence, namely, redness, oedema, and swelling of the soft parts covering the mastoid process with a consequent forward, downward, and outward displacement of the auricle, best seen when the patient's head is viewed from behind. When, on the other hand, the abscess is forming in the deeper regions of the mastoid area, and especially when a thick cortex happens to exist, there may be absolutely no external indication of its presence, the main symptoms being deep-seated pain and profuse discharge from the middle ear. As the outcome of a collection of pus in the mastoid cells, more especially in those abutting against the posterior meatal wall, there is swelling of the superimposed soft tissues causing stenosis of the meatus.

Examination of the membrana tympani shews as a rule bulging of its posterior segment with frequently a small conical perforation, or perforation of Shrapnell's membrane. The amount of discharge varies greatly; at times it is profuse and almost continuous; at other times it is almost absent even while the disease within the mastoid is making progress. In exceptional cases the discharge from the middle ear may cease entirely and the perforation may cicatrise, the mastoid empyema suddenly lighting up weeks afterwards.

Facial paralysis or paresis is not common in acute middle-ear or antral suppuration. It, however, sometimes occurs in cases of tuberculous, exanthematous, or influenzal origin.

Terminations.—In certain circumstances an empyema of the mastoid cells may undergo spontaneous resolution; more usually, however, a gradual breaking down of the cancellous tissue of the process takes place, with ultimate erosion of the cortex and formation of a fistulous track leading into a subperiosteal abscess, or a perforation of the posterior meatal wall or medial mastoid table. Sometimes pus tracks backwards through the posterior mastoid wall, opens up the sigmoid groove, and ends in the formation of a parasinuous abscess with or without thrombosis of the sinus. In other cases the antral roof becomes eroded and perforated, leading to intracranial lesions, such as meningitis, and extradural, cerebral, or cerebellar abscess. In rare cases the outer labyrinthine wall becomes perforated, followed by acute suppurative labyrinthitis.

Prognosis is as a rule favourable and depends mainly upon the underlying cause and the duration of the disease. Generally speaking, the earlier the purulent focus is opened and drained the more rapid the healing, whilst the longer the disease lasts the greater the destruction of adjacent tissue and the greater the risk of extension to vital organs. The prognosis is also better in simple pathogenetic infections than in those following the exanthemata, enteric fever, tuberculosis, and other infections.

Treatment.—In the early stages of acute inflammation of the mastoid cells resolution may frequently be brought about by securing free drainage from the cavity of the middle ear, by local depletion, or by the local application of cold combined with rest in bed in a warm room and the internal administration of saline aperients. To obtain efficient drainage from the infected tympanic cavity an intact membrane should be freely incised either horizontally or vertically, whilst a small perforation should invariably be enlarged so as to secure a free and uninterrupted egress for all inflammatory products. Local depletion is best effected by the application of from four to six leeches, or scarification with Heurteloup's artificial leech. Cold may be applied either by means of suitably shaped ice-bags or Leiter's apparatus. Counter-irritation over the mastoid process and inflation of the middle ear either by Politzerisation or catheterisation should be avoided.

If antiphlogistic treatment of this kind fail after a trial of from three to four days operative interference should be undertaken at once. It is better, when any doubt exists, to operate at an early stage of the disease than to allow progressive destruction of the contents of the tympanic cavity and mastoid area to proceed unchecked. Whether complete ablation of the mastoid cells and opening of the mastoid antrum at the same time are necessary must be decided by what is found after removal of the mastoid cortex. In many cases of acute mastoid suppuration the abscess is subcortical and localised and is readily cured by simple drainage. On the other hand the abscess may occupy the greater portion of the cellular area of the process and antral cavity and demand a complete clearing out of all cancellous tissue.

Operation for Acute Suppurative Endomastoiditis.—An incision is made ¼ inch behind and parallel to the attachment of the auricle from the base of the mastoid process to its apex. The soft tissues are divided down to the bone along the whole length of the incision, bleeding points ligated, and with a periosteal elevator the periosteum is pushed forwards—in one sheet, if possible—until the posterior border of the bony meatus is fully exposed. In a similar manner it is pushed backwards until the whole process is completely bared. By means of a gouge and mallet, or a hand-gouge, the cortex is carefully removed until the subcortical mastoid cells are fully exposed. All softened bone, granulation-tissue, and purulent debris are removed, the whole of the interior of the process being cleared out if necessary. In this way the mastoid antrum and all adjacent and infected cells are surgically cleansed or removed.

After-treatment.—The cavity thus formed may be allowed to fill up with granulation-tissue, light packing being used as a dressing, or, in suitably selected cases, it may be allowed to fill up with blood-clot which organises and induces a rapid healing. If the "blood-clot" method of treatment be adopted no stitches should be inserted.

Influenzal Mastoiditis.—One of the peculiarities of post-influenzal suppurative middle-ear disease is the intensity and acuteness of the inflammatory process and its tendency to invade the mastoid cells, more especially those situated towards the apex. The inflammatory process is accompanied by great pain and by rapid disintegration of the cellular spaces of the mastoid process. The result is the formation of a subcortical mastoid abscess, usually apical.

The *clinical manifestations* of a subcortical mastoid abscess are localised pain much increased by pressure, especially by pressure upon the anterior wall of the mastoid apex, by a sagging of the postero-superior meatal wall, and subsequently by localised mastoid periostitis.

In the *treatment* of such cases the subcortical abscess should be opened by a free removal of the mastoid cortex and all softened bone and succulent granulation-tissue scraped out with a sharp spoon.

It is held by many that in such cases it is unnecessary to open and drain the mastoid antrum, but as the track of infection is by way of the antral cavity, it would appear to be wiser to lay open the antrum and all adjoining mastoid cells as far as the terminal apical cells, preserving the cavity of the middle ear and "iter ad antrum" after having freely enlarged the existing perforation in the membrane so as to secure good drainage. Such cases heal remarkably well, the cavity in the bone filling up with healthy granulations, which subsequently contract and leave a dense and firm cicatrix.

Suppuration of the Middle Ear and Mastoid Cells in Enteric Fever.—Purulent inflammation of the middle ear occurring during enteric fever is not particularly common. When it does occur it usually takes place during the fourth or fifth week of the disease, and is due either to extension of inflammation from the pharynx or naso-pharynx, or to embolic abscesses in the tympanic mucosa secondary to some purulent focus elsewhere in the body. The resulting perforation of the membrana tympani is as a rule fairly large, and is situated in its posterior segment. In many such cases complete healing of the perforation takes place with recovery of hearing power. On the other hand, as the result of extension of the inflammatory process to the peripheral fibrils of the auditory nerve, or as the result of central implication of the nerves, severe if not complete deafness may follow. In other cases extension may take place towards the mastoid area, or, as the outcome of perforation of the roof of the middle ear or antrum, intracranial suppuration may be induced.

Treatment.—If the general condition of the patient permit, cleansing of the naso-pharynx is an excellent prophylactic. This may be accomplished by a spray or douche, or by the local application of some antiseptic pigment. In the event of the middle ear becoming infected, free drainage should at once be secured and antiseptic treatment instituted.

Bezold's Mastoiditis is characterised by perforation of the median wall of the mastoid process and by the formation of an abscess under the deep cervical fascia. Circumstances favourable to its development are the presence of a pneumatic mastoid process with large thin-walled apical cells, and of a cortex of sufficient thickness to offer resistance to the exit of pent-up purulent exudation. Once perforation of the inner mastoid wall has taken place tracks of infection spread along the sheaths of neighbouring muscles inwards towards the pharynx—retro-pharyngeal abscess—or downwards to the anterior mediastinum—depression-abscesses. In exceptional cases pus may track under the base of the skull, causing ultimate necrosis of the occipital bone.

The *symptoms* are pain on pressure over the mastoid apex, localised oedema in the same situation, and the formation of a deep-seated swelling in the neck, at first hard and brawny, but gradually becoming softer as the pus works its way to the surface. Such cases are not infrequent as the result of an influenzal otitis media. The course of the disease is apt to be protracted on account of the burrowing of pus within the deep tissues of the neck, and the difficulties met with in securing efficient drainage.

Treatment.—Complete ablation of the mastoid cells is necessary. All foci of softened bone and oedematous granulation-tissue are to be cleared out with a sharp spoon down to the tip of the mastoid process, the apex itself being removed if necessary. The fistulous track upon its inner wall is to be enlarged, and a blunt-pointed probe passed into the neck and made to project externally at the lowest level of the abscess cavity. The probe is then cut down upon from the outside, the contents of the abscess evacuated, and a drainage-tube drawn upwards to the floor of the cavity already made by the removal of the apical cells. The abscess cavity is irrigated daily until all secretion of pus has ceased, when the tube is withdrawn. The cavity within the mastoid is packed from time to time until it becomes filled up with healthy granulation-tissue.

Diseases of the Mastoid Antrum and Mastoid Cells in the course of Chronic Suppurative Otitis Media.—In all chronic suppurative affections of the middle ear the posterior end of the middle-ear cleft becomes affected. Whether symptoms of urgency arise or not depends upon the presence or absence of factors contributing to the retention of secretion, or upon the incidence of intercurrent attacks of acute inflammation. The extent of implication of the mastoid cells depends also to a large extent upon the anatomical structure of the mastoid process, upon the duration of the previously existing intra-tympanic disease, and upon the general health of the patient.

The actual development of progressive local changes in the mastoid antrum hinges to a considerable extent also upon the existence of polypi or granulation-tissue within the middle ear, the presence of cholesteatoma, the existence of epitympanic disease with a small perforation in Shrapnell's membrane, intra-tympanic adhesions, stenosis of the external meatus, and so forth. These various pathological conditions, by causing retention of secretion, favour infection of the mastoid cells and the formation of a secondary suppurative endomastoiditis.

The *pathological changes* which ensue consist, in the first place, of a thickening of the mucosa as the result of an interstitial hyperplasia, obliteration of the smaller mastoid cells, formation of new bone with a consequent osteosclerosis, or degenerative changes in the form of purulent collections, caries, necrosis, or a cholesteatoma. The main site of these changes is in and around the mastoid antrum itself, the vertical cells both in the pneumatic and diploic processes being much less frequently affected. As the result of chronic suppuration within the mastoid area the whole of the cancellous tissue may be eaten away, its place being taken by a collection of pus, broken-down purulent debris, sequestra, and so forth. Even the dense bone forming the mastoid cortex may become gradually eroded, with the result that perforation of the outer table with the formation of a subperiosteal abscess ensues, or perforation of the roof or posterior wall with consequent escape of pus into the middle or posterior fossa. In tuberculous, syphilitic, and diabetic subjects the greatest destruction of bone takes place, so much so that even at a comparatively early stage of the disease a mere shell may be left enclosing a highly infective abscess cavity. At any moment the existing endomastoid disease may become lit up, with the result that grave symptoms suddenly supervene. On the other hand the disease may remain latent for years, the only symptom of any consequence being the presence of an intractable and foul-smelling discharge from the middle ear. The presence of chronic suppurative bone disease in and around the middle ear is, however, always fraught with grave danger to the patient. Perforation may take place suddenly with subsequent acute intracranial infection, or a generalised infection may ensue, manifesting itself in an attack of acute septicaemia or pyo-septicaemia.

Symptoms.—So long as no actual retention of secretion takes place, and so long as no intercurrent inflammatory attack is grafted on to the underlying chronic process, the disease may last for years. If, on the other hand, the previously dormant infective process becomes lit up as the result of retention of secretion, or by bacterial infection following some acute disease or injury, severe symptoms at once supervene. Pain over the region of the mastoid antrum, aggravated by pressure, is at once complained of; there is a rapid rise of temperature accompanied by quick pulse, whilst tinnitus, vertigo, lassitude, and sleeplessness are frequent. If any cerebral hyperaemia coexist, vomiting is usual. Should the disease originate in the deeper portions of the process pain is all the more severe, whilst external evidences of its presence, such as redness, oedema, and so forth, are slow in appearing. On the other hand, should the main focus of infection be comparatively superficial, there is at an early stage swelling and oedema of the superimposed mastoid tissues, with possibly the formation of a subperiosteal mastoid abscess, and a marked downward, outward, and forward displacement of the auricle. The canal of the external auditory meatus is as a rule stenosed, as the result of swelling of its postero-superior wall, whilst the membrana tympani, which is in consequence largely hid from view, may be swollen and covered with granulations. Should the disease go on unchecked, perforation of the cortex usually takes place. In rare cases the posterior meatal wall gives way with the formation of a fistula leading into the depths of the mastoid process. In certain instances, especially where a large apical pneumatic cell exists in conjunction with a thick cortex, perforation of the median mastoid wall ensues with the formation of an abscess under the deep cervical fascia (Bezold's mastoiditis).

In the *treatment* of acute infective conditions grafted upon an existing chronic suppurative basis surgical interference is as a rule imperative. Conservative local treatment is usually of comparatively little value; it is, however, indicated where the intercurrent inflammation is of no great severity, where there is no serious interference with drainage, and where a cytological examination of the discharge reveals but few lymphocytes and no myelocytes. In such cases rest in bed, the local abstraction of blood, the use of Leiter's cold coil, and the frequent irrigation of the meatus with warm alkaline antiseptic lotions may serve to arrest the progress of the inflammatory process. Local treatment is contra-indicated in cases in which recurring attacks of acute inflammation are common, in which there is deep-seated caries or evidence of cerebral irritation, and in which an examination of the discharge reveals the presence of numerous myelocytes or of acid-fast squames pointing to the presence of a cholesteatoma. In all such cases some form of operative interference is necessary; the particular form of operation depends upon the extent of the disease, the amount of destruction already existing within the middle ear, and the degree of audition still present. By means of suitable surgical interference it is possible not only to arrest the existing local disease, but to free the patient of all risks of secondary intracranial infection, and in many instances to improve the existing degree of hearing power.

The Schwartze operation, which formerly was universally performed in such cases, has now been practically abandoned on account of the difficulties encountered in maintaining free drainage and the impossibility of getting rid of all diseased bone, etc. Its place has been taken by the operations planned by Küster, Zaufal, and Stacke, which are now described as the "radical operation." The aim and object of all operations of a radical nature performed in cases of chronic suppurative bone disease in and around the middle ear is to throw the tympanum, the mastoid antrum, the attic, and the external auditory meatus into one large space with smooth walls, and ultimately to bring about complete epithelialisation of the cavity so produced.

The indications for opening up the various accessory cavities of the middle ear depend upon certain subjective and objective clinical phenomena. As a rule both sets of symptoms are present, and taken together make up the clinical picture necessitating operative interference. The main symptoms are the occurrence of continuous or frequently recurring pain, at times local, at other times radiating over the affected side of the head, and evidence of impending cerebral mischief such as vomiting, vertigo, and impaired movements of the head. The principal objective indications are:—(1) The presence of an intractable purulent and fetid discharge from the middle ear which is uninfluenced by local and minor surgical measures, and which on bacteriological examination is found to contain large numbers of streptococci or meningococci, and on cytological examination numerous lymphocytes, myelocytes, or acid-fast squames. (2) Deep-seated caries of the temporal bone. (3) Polypi or bone granulation-tissue constantly recurring after removal. (4) Fistulous openings either through the mastoid cortex or posterior meatal wall leading down to deep-seated foci of caries. (5) Deep-seated cholesteatoma. (6) The occurrence of facial paralysis during the course of chronic suppurative middle-ear disease. (7) Repeated attacks of acute mastoiditis grafted upon a chronic basis. (8) Tuberculous disease of the middle ear or adjacent mastoid area. (9) Stenosis of the external auditory meatus interfering with the exit of pus from infected and deep-seated areas.

In determining which particular form of operation to adopt the surgeon must be guided by the extent of the disease, so far as it is possible to make this out, and by the amount of hearing power still remaining. In cases in which the hearing power is fairly good, it is advisable if possible to conserve the remains of the membrane and the ossicular chain. On the other hand where great and irreparable damage has already been inflicted upon the contents of the middle ear, and where the hearing power is seriously affected, no particular object is gained by attempting to retain either the remnants of the membrane or the ossicular chain. In order to obtain access to the accessory cavities of the middle ear, while at the same time preserving the tympanic contents, Küster (1889) suggested removal of the posterior meatal wall, followed by a toilet of the antrum and any adjacent infected mastoid cells. In 1890 he adopted von Bergmann's plan of opening up the squamous cells of the roof of the meatus. This procedure was followed by the operations designed by Stacke and Zaufal. In the Stacke operation the auricle and cartilaginous meatus are detached, and the attic and antrum opened up by removal of the postero-superior meatal wall, whilst in the Zaufal operation the anterior part of the mastoid process and the postero-superior portions of the meatal wall are first cut away, and subsequently the attic and antrum are opened.

Stacke's Operation.—An incision is made behind and parallel to the attachment of the auricle, commencing at the tip of the mastoid process and continued forwards into the temporal region. The soft tissues, including the periosteum, are now drawn forwards so as to expose the superior and posterior margin of the bony meatus freely. With a small raspatory the cartilaginous meatus is separated from its attachments to the superior and posterior bony walls of the meatus and cut through as near to the attachment of the membrane as possible. The auricle and the detached cartilaginous meatus are now drawn forward and held forward by means of a rake or Allport's retractor (Fig. 56) or suitably disposed piece of bandage. All remains of the membrana tympani are now removed, together with the malleus, if still present. A Stacke's protector is introduced into the attic and its external wall cut freely away until the roof of the middle ear and the superior wall of the external meatus form one continuous plane. The incus, if still present, is extracted with fine forceps and the protector pushed gently backwards along the "iter ad antrum." All bone forming the postero-superior wall of the meatus is chiselled away until the protector enters the antrum quite freely. Finally all bone external to the cavity of the antrum is chiselled away until antrum, tympanum, and external meatus form one continuous cavity. It is important that so much of the posterior meatal wall be removed as to admit of the inferior wall of the auditory meatus passing directly into the inferior wall of the antrum. All diseased mucosa, carious bone, and cholesteatomatous debris is thoroughly scraped out with a curette, the walls of the cavity carefully smoothed by hand- or electrically-driven burr, and flaps made from the cartilaginous meatus to line as much of the bared bony walls of the cavity as is possible.

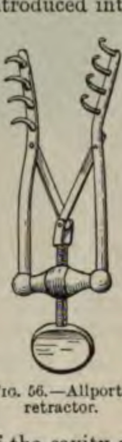
FIG. 56.—Allport's retractor.

The "complete" radical mastoid operation is indicated in cases of chronic suppurative middle-ear disease in which, after prolonged and careful antiseptic treatment, purulency continues unchecked, in which there is an obvious deep-seated bone lesion not amenable to local medication or minor surgical measures, and in which the contents of the middle ear have been seriously damaged by continuous suppuration. A careful cytological examination of a droplet of discharge taken from the depths of the middle-ear cleft is of much value in settling the question of operative versus non-operative treatment. Where the discharge is found to contain numerous lymphocytes and myelocytes there need be no hesitation in advocating operative interference, and where the middle ear has already been almost entirely destroyed by the ravages of the suppurative process and the sense of audition irreparably damaged the performance of the complete post-aural or Schwartze-Stacke operation is indicated, and is almost invariably followed by an improvement in the general health of the individual and, as a rule, by an increased amount of hearing power.

The auricle having been thrown forward, as already described, and the posterior bony margin of the external meatus brought clearly into view, the cavity of the mastoid antrum is freely opened, care being taken to slope the walls of the cavity so as to produce a conical-shaped opening with its truncated apex at the deepest part of the mastoid antrum. The cartilaginous meatus is now completely separated from its bony connexions to the posterior and superior meatal walls, and drawn forwards by means of a retractor or strip of bandage. The remains of the membrana tympani and ossicula auditus are now clearly visible at the bottom of the meatus. An antrum hook is passed along the "iter ad antrum," and held in position by an assistant while the operator removes by means of chisel, gouge, or bone forceps the bridge of bone separating the tympano-meatal from the antral cavity. This bridge of bone must be so thoroughly cut away that when a bent probe is introduced along the roof of the cavity there is no obstruction of any sort met with during its withdrawal. The bridge should also be cut away with a gentle upward slope from without inwards so as to avoid injury to the facial nerve in its passage towards the stylo-mastoid foramen (Fig. 57). The remains of the membrana tympani, the malleus, and the incus, if present, are now carefully detached by forceps or curette and the outer attic wall removed. In this way the auditory meatus, the tympanum, the mastoid antrum, and any contiguous mastoid cells which have been cleared out during the course of the operation are thrown into one large cavity—the antro-tympanic cavity—the walls of which must be carefully smoothed down by hand or electric burr so as to present no rugosities for the retention of secretion. As the ultimate aim of the operative procedure is to obtain a cavity lined by healthy epidermis, use is made of the cartilaginous meatus to assist in covering a portion at least of its bony walls. With this end in view flaps are fashioned from the cartilaginous meatus according to the methods of Panse, Körner, Stacke, Siebenmann, Ballance, and the author. In order to cut the Panse flap a knife is introduced into the meatus and the posterior cartilaginous wall divided along its middle line and as far outwards as the concha. At this point another incision is made at right angles to the first, both upwards and downwards, so that two quadrilateral flaps are produced, an upper and a lower. These flaps—when the auricle is ultimately replaced—are kept in position either by stitches or by packing introduced through the meatus. From the edge of these flaps epithelium creeps slowly over the bony walls of the antro-tympanic cavity, carpeted as it is at this stage with a layer of healthy granulations. The flap suggested by Körner is fashioned as follows: an incision is made through the cartilaginous meatus at the line of junction of its superior and posterior walls and another at the line of junction of its posterior and inferior walls. These incisions are carried outwards well into the concha. The quadrilateral flap thus formed is folded backwards and made to cover a portion of the posterior wall of the antro-tympanic cavity. By the bending backwards of the incised concha the auditory meatus is thereby somewhat enlarged and a better view of the recesses of the cavity obtained. Körner's

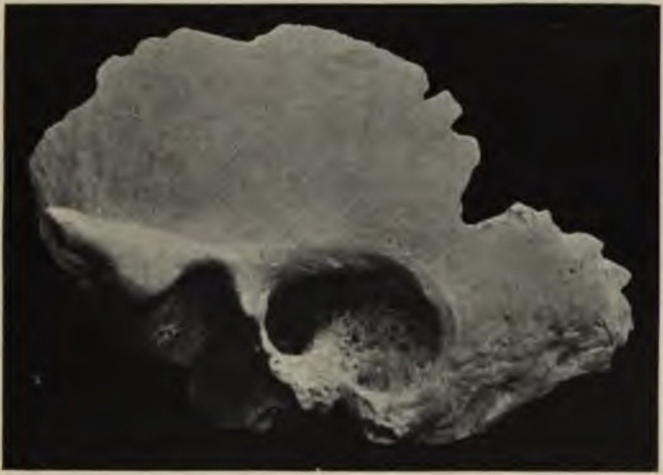

FIG. 57.—Complete post-aural operation.

flap is especially useful when the post-auricular wound is closed at the time of operation, Panse's when it is advisable to preserve post-auricular drainage.

In Stacke's plastic operation a rectangular flap is obtained by making a longitudinal incision through the superior wall of the meatus as far outwards as the concha, and a vertical incision through the concha at right angles to the former. The flap thus formed is folded downwards and backwards and kept in position by means of packing introduced through the meatus.

The author's flap is made by incising the cartilaginous meatus along the line of junction of its superior and posterior walls well outwards into the concha. By sweeping the knife round in a circular manner the concha is divided parallel to the antihelix as far downwards as the floor of the meatus. The crescentic flap thus formed is folded downwards and backwards against the floor of the cavity in the bone, and kept in situ by two sutures passed from without through the tissues of the neck and through the flap and tied over a rubber tube. In this way a considerably enlarged meatus is produced, an advantage alike for inspection and subsequent dressing.

In all these plastic operations it is advisable to cut away as much redundant soft tissue as possible, so that the flaps may lie accurately against the bony walls of the cavity. At the same time it is essential not to interfere with the nutrition of the flaps for fear of subsequent sloughing or gangrene.

To facilitate the lining of the cavity the implantation of epithelial grafts has been suggested, and is now extensively adopted. Ordinary Thiersch grafts are cut and placed upon the granulating walls of the cavity. Should the granulating surface be aseptic, the grafts, as a rule, take, and the process of epidermisation is materially hastened. The method of skin grafting suggested by Mr. C. A. Ballance is of much value both in securing complete epidermisation and in hastening the healing process. In the initial stage of the operation Mr. Ballance makes a crescentic incision parallel to the attachment of the auricle and along the hair line. The soft parts are dissected from the underlying bone and the flap containing the auricle and the auditory meatus drawn well forwards. The opening of the antrum and the accessory sinuses is conducted as in the Schwartze-Stacke operation. The special flap taken from the soft parts is constructed as follows: the inferior meatal wall is cut through along its entire length and well into the concha. The incision in the concha is carried upwards and backwards in a curved direction as far as the crus helicis. The flap after having been trimmed of all superfluous soft tissue is folded backwards and fixed by means of three or four sutures to the original mastoid flap. The external incision is now closed by stitches. After the lapse of from seven to ten days the wound is reopened and carefully cleansed with sterilised normal salt solution. The walls of the cavity will at this stage be found to be covered by a delicate layer of pink granulations, which, if aseptic, afford an admirable soil for the implantation of a skin graft. A large graft is cut from the front of the patient's thigh or from the outer surface of his arm and floated from sterile normal salt solution on to a special lifter (Fig. 58). From this lifter it is gently insinuated into the mastoid cavity and made to cover as much of the granulating surface as possible. By a combination of suction and pressure the graft is driven home so as accurately to line the large antro-tympanic cavity. Formerly it was customary to cover the graft with pure gold leaf so as to prevent the dressings from pulling it out from the cavity subsequently; at the present time most operators are content to keep it in position by means of small pads of sterilised wool or strips of gauze. The auricle having been replaced, the post-auricular wound is resutured. At the end of five or six days the dressings are removed, when it will usually be found that the graft has taken. Superficial layers of the graft become detached and come away in the dressings, the main body of the graft, however, remaining accurately in situ. Successful grafting of the antro-tympanic cavity very materially hastens the after-treatment and frees the patient from the painful process of packing.

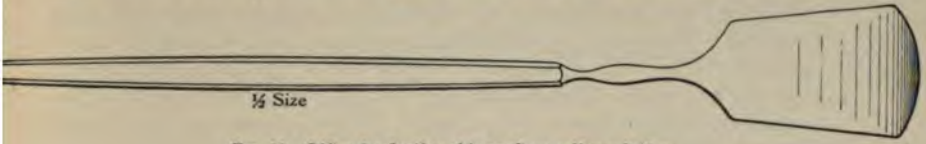

FIG. 58.—Lifter for floating skin graft on saline solution.

After-treatment of Operation Cases.—In the after-treatment of mastoid operation cases it is desirable to keep the patient in bed for at least ten days. By so doing the process of repair is materially hastened and the granulation-tissue formed is healthier and more robust. After the flaps have been fixed in position and the antro-tympanic cavity thoroughly cleansed, packing should be introduced so as to fill the cavity and to exert a gentle and uniform pressure upon its walls. By so doing the flaps derived from the cartilaginous meatus are also kept in accurate apposition against the bony walls of the cavity. This packing should remain in situ for from four to six days. It is advisable to change all external dressings every second or third day. At the end of the fourth or fifth day all packing should be gently drawn out. This is much facilitated by soaking it first of all with peroxide of hydrogen solution. The antro-tympanic cavity may now be cleansed with some warm alkaline antiseptic solution or wiped dry with sterilised wool. There is considerable difference of opinion whether packing should be reintroduced or whether treatment by instillation or antiseptic lotions should be practised. The objections to continuous packing are that it is painful, that it tends to keep the cavity in a sodden condition, and that the granulations springing from the bone tend to be pale and weakly and less able to support the epithelium which is slowly creeping over the walls. The advantages of fluid instillations are their painlessness and their stimulating effect upon the growing epithelium from the cut edges of the flaps. The object of all after-treatment being to encourage the "papering" of the granulating walls of the cavity with healthy epithelial cells, the instillation method of treatment will, as a rule, be found the most serviceable and useful. Should treatment by packing be decided upon, it is advisable to dress the cavity every third or fourth day. On the other hand, the after-treatment by instillation of alkaline antiseptic solutions should be carried out at least twice daily.

As a rule the process of after-treatment, even in quite favourable circumstances, occupies a period of at least two months, and frequently considerably longer. The main difficulties encountered are the keeping of the cavity in an aseptic condition, and preventing the formation of exuberant granulation-tissue. Exuberant granulations should be kept down by curetting or the application of chromic acid, nitrate of silver, or persulphate of iron. The more aseptic the cavity is maintained the quicker the process of epithelialisation. The inner tympanic wall is the most difficult part to line with squamous epithelium, and in many cases always remains unlined. Whether the external wound should be completely closed or not at the time of operation depends upon the condition of the wound, the extent of the operation, and upon whether all disease has been completely cleared out or not. In cases in which a comparatively small area of bone has been removed there is no reason why the wound should not be closed at once and subsequent dressings conducted through the meatus; on the other hand, where the disease has been very extensive or deep-seated, or where a cholesteatoma has been present, it is inadvisable to close it in the first instance at any rate. In the event of a cholesteatoma having been present, it is held by many competent otologists that the maintenance of a permanent post-auricular fistula is desirable. The closure of the external wound at the time of operation has the further advantage of allowing the patient to dispense with bandages at a very early stage of the after-treatment. The ultimate result of all successful post-operative treatment should be to produce a cavity completely lined by a highly refractive dry squamous epithelium. Should the fluid method of after-treatment be adopted, watery or alcoholic solutions of boracic acid, perchloride of mercury, or peroxide of hydrogen will be found serviceable. These solutions should be retained in the cavity—after previous cleansing—for from 10 to 20 minutes twice daily, and the external meatus subsequently plugged with boracic or iodoform wool.

A difficulty sometimes encountered is the tendency to stenosis of the meatus. This, however, should not happen if the flaps have been properly cut, and if they have been maintained in accurate apposition against the bony walls of the cavity by efficient packing or by suitably disposed sutures. To assist in preventing collapse of the meatal walls a large rubber tube is sometimes inserted into the meatus. Its presence is, as a rule, however, very uncomfortable, and tends to produce sloughing of adjacent soft tissues.

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W. M.

THE INTRACRANIAL AND INTRAVENOUS INFECTIONS COMPLICATING EAR DISEASE

By Charles A. Ballance, M.V.O., M.S.

The occasional association of fatal intracranial suppuration with discharge of pus from the ear was certainly known in Greek and Roman times, and perhaps even earlier, but to Morgagni belongs the credit of having first demonstrated the pathological relationship between the two affections. Before his time, even by his immediate predecessor, Valsalva, the intracranial suppuration was held to be, in most, if not in all cases, the primary disease, and the aural suppuration the secondary. Our great masters of clinical medicine, such as Abercrombie, Bell, Bright, and Watson, described and recognised these cases, but realised no important progress either in preventive or curative treatment; nor did Wilde and Toynbee, greatly as they added to our knowledge of diseases of the ear, do much more. Surgical intervention, so ably advocated by Jean Louis Petit, the justness of whose views was for long unappreciated, is now carried out with constantly increasing precision and success, even when the disease has extended beyond the limits of the temporal bone. The probability of cure by an appropriate operation renders the differential diagnosis of the intracranial complications of ear disease not only of clinical interest, but of vital importance.

The conditions coming within the scope of this article may be grouped as: (1) *extradural suppuration*; (2) *venous infection*: (*a*) thrombosis, simple or infective; (*b*) systemic septic intoxication (sapraemia); (*c*) systemic infection of septicaemic or pyaemic type; (3) *intradural infection*: (*a*) meningitis serosa; (*b*) subdural suppuration, localised or diffused; (*c*) subarachnoid suppuration, localised or diffused; (*d*) brain abscess; (4) *tuberculosis*. Any one, or almost any possible group, of these conditions may result when infective material makes its way from within the temporal bone to the interior of the cranial cavity.

Pathology.—It will be convenient to discuss the pathology under the following headings: (1) brain abscess; (2) venous infection; (3) meningeal infection; (4) tuberculosis.—1. *Brain Abscess.*—In most cases there is a track of caries through which pus has passed from the focus of disease in the temporal bone to the interior of the skull. Other, less obvious routes, occasionally followed by the infective material, are: (i.) by way of a congenital defect in ossification (dehiscence); (ii.) by way of one of the prolongations of dura mater which are embedded in the temporal bone; (iii.) by way of the labyrinth or one of the bony channels for the passage of vessels and nerves. Having reached the dura, the pus sets up inflammation of this membrane, and the first stage of intracranial inflammation is established. The dura may offer great and prolonged resistance, and a more or less considerable abscess between the bone and the dura be the sole result. Sooner or later, however, pus generally makes its way through the inflamed dura and reaches the arachnoid and pia; occasionally diffuse suppuration is set up in the subdural or subarachnoid space with fatal result, but commonly adhesions have taken place before the dura is perforated, thus limiting the spread of infection in the meninges, and a suppurating track leads on into the brain. The cortex is very vascular, and its connective tissue, reinforced by numerous prolongations of the pia mater, is abundantly provided with cells; it is therefore well able to resist bacterial invasion, and commonly suffers but little destruction. The lymphatic sheaths of the numerous blood-vessels which traverse the cortex at right angles to its free surface afford a ready passage into the white substance beyond. Where the infection traverses the cortex a barrier of newly-formed connective tissue limits the lesions to a narrow track, but the white substance, much less resistant, is more or less extensively destroyed, so that a pear-shaped abscess cavity is formed, with a narrow stalk passing through the cortex and adherent to the bone at the spot where the disease found entrance. This is the form of otitic brain abscess most frequently met with. Once started, abscess in the brain may spread without intermission, and burst, either into the ventricles or on the surface, with rapidly fatal result; or it may run a chronic course, with more or less complete latency, so far as symptoms are concerned. In such cases the abscess cavity becomes limited by a fibrous capsule, which may be so complete and dense that the abscess can be shelled out whole. According to Körner, most otitic brain abscesses are encapsuled. The communication with the diseased bone through the stalk may become obliterated, and the stalk become reduced to a fibrous band. The earlier the abscess is seen, the nearer will it be found to the spot where the disease first penetrated the bone, and the more evident will be the stalk or its remains. Such are the main points about the gross morbid anatomy of cerebral abscess by direct extension of disease from the bone to the brain.

But here, as elsewhere, infection may be carried by the blood-stream, and so a focus of suppuration arise in the brain at a considerable distance from, and without obvious connexion with, the primary focus in the bone. Such an abscess, like the abscess from direct extension, may spread continuously until it ruptures, or may run a chronic course and become more or less completely encapsuled. Encapsulation is no bar to the further extension of an abscess, nor even to its rupture into the ventricles. Abscess is not the only lesion of the brain caused by infection from the temporal bone; diffuse encephalitis, quite comparable to diffuse cellulitis, of a limb, may result,—a rapidly fatal lesion. Moreover, around an abscess there is often an area of encephalitis and haemorrhagic softening, an occasional cause of persistence of symptoms after operation.

2. *Venous infection*, another great danger incidental to infective disease of the temporal bone, arises chiefly by the disease spreading by direct continuity into the groove for the sigmoid sinus, though other sinuses

may be primarily infected, and infection may, as elsewhere, occur from septic thrombosis of the small veins in the bone itself. The first effect of infection of the sinus by pus entering the groove is thrombosis. A great venous channel, such as the sigmoid sinus, is not suddenly occluded by thrombosis. The clot which first forms in the vessel lies along its wall, but does not occlude its lumen. By the addition of successive layers, the clot may extend so as completely to arrest the stream of blood. Blocking of the channel may, however, never occur in consequence of the cessation of coagulation, or a block once formed may not persist on account of contraction of the clot. The clot, for a time which varies with the virulence of the infection, remains uninfected, but unless the primary source of infection is promptly suppressed, the infective material makes its way through the wall of the sinus and invades the clot, so that there comes to be an additional local centre of infection continuous with the primary one, from which not only micro-organisms, but gross particles of infective clot may be carried into the circulating blood. The infected clot breaks down and suppurates, the outer wall of the sinus sloughs, and pus within the sinus communicates freely with the pus outside. Such an abscess, though intravenous, is not intradural, and does not become so unless the inner wall of the sinus sloughs. Clot forms in the sinus above and below, and may for a time limit the infection to a particular segment of the sinus, the condition then being a local intravenous abscess; in such cases, as long as the clot isolating the abscess is uninfected and occludes the vessel completely, there is no danger of general infection by way of the venous channel, but the risk of meningitis, or brain abscess, as a further complication is rather increased than otherwise. When, however, the stage of intravenous suppuration is reached, the barrier is in most cases inefficient, and then there arises that most dangerous condition, an infective thrombus with a but partially obstructed blood-current. Infection extends both towards the heart and towards the torcular. The cavernous sinus may be infected by way of the petrosal sinuses or the carotid sinus, or directly by extension of bone disease in the cellulae petrosae to the apex of the petrous. The opposite cavernous sinus often becomes infected through the circular sinus. On the right side the clot extending backwards may reach the superior longitudinal sinus, and when the communication at the torcular between the two lateral sinuses is abnormally free, the thrombus may extend across the middle line to the opposite sinus. The jugular bulb, when infected, commonly is so by extension from the sigmoid sinus, but venous infection may commence in the bulb itself, the pus reaching it through a carious erosion or congenital dehiscence in the floor of the tympanum: cases in which this occurs are commonly acute, and without a long previous history of otorrhoea. Infected clot in any part of the sinus or jugular vein soon leads to general septicaemia, or to pyaemia with metastatic abscesses. Particles of uninfected clot are occasionally detached and carried along with the blood-stream as emboli. These determine simple infarction in lung, kidney, spleen, or wherever they happen to lodge.

The special facility with which, on account of their anatomical relations, the large venous sinuses become infected in temporal bone disease does not exclude the possibility of pyaemic infection through the small veins of the bone itself, which used to be so common a feature of infective bone disease elsewhere, and so we occasionally see pyaemic infection from temporal bone disease without infection of the sinus. In such cases the micro-organisms, as Körner points out, not being enclosed in gross particles of clot, are able more easily to traverse the pulmonary capillaries, and hence pulmonary metastases are a less common feature of such cases. Mural thrombi in the jugular bulb may sometimes be overlooked at a necropsy. When they are present, and become infected, they constitute an insidious and dangerous complication. The collapse of the jugular vein from obstruction of the bulb and sigmoid sinus may be so complete that when exposed by operation it appears as a cord, and the operator, unless aware of this, may be embarrassed.

3. *Meningeal Infection.*—In most cases of intracranial infection from ear disease the meninges are only slowly reached by the infection, and, moreover, the dura itself offers prolonged resistance to perforation. It is well known that when pus slowly makes its way to a serous membrane adhesion of the two layers takes place, and if the infection proceed farther it traverses both layers without causing general infection of the cavity. In the pleura and peritoneum the serous surfaces are kept in constant lateral movement, and infective material is rubbed over a considerable area before adhesions can take place. In the arachnoid the mechanical conditions are different, there being no appreciable lateral movement. The two layers therefore can, and commonly do, become adherent before any considerable area is affected, hence any collection of pus between dura and pia is commonly quite small in amount. Sometimes, however, infection reaches the arachnoid and pia before adhesions have taken place, and then instead of forming a mere track through these membranes, and passing on to the cerebral substance, a diffused or localised meningeal inflammation results. In the skull, as elsewhere, the disease may be arrested in the serous stage. Inflammation of the pia mater is neither clinically nor anatomically distinguishable from inflammation of the arachnoid, but either the subdural or the subarachnoid space may be the exclusive or the chief seat of the inflammatory exudation, a point not without significance in the treatment. Diffuse suppuration in the subdural cavity is uncommon except as the result of direct infection by injury, but I have seen it occur in otitis and in influenza. Certain varieties of pus seem to have but little tendency to perforate serous membranes (such as the arachnoid or peritoneum), and but little irritant effect upon them. The pus may be spread out in a sheet of greater or less thickness over a certain limited area of the visceral arachnoid, though there may be no visible adhesions present which have checked its spread. The more rapidly the dura is traversed by infection the greater the danger of diffuse suppurative meningitis. In infective injuries of the head meningitis is much more common than brain abscess,

but in ear disease brain abscess is a more frequent complication than diffuse suppurative meningitis. In those cases, now happily very rare, in which intracranial inflammation has followed unskilful attempts to remove a foreign body from the ear, meningitis has been the usual result, the causative condition being an injury rapidly opening up the way to the meninges, and not a disease making its way slowly through the bone.

Another way in which a fulminating meningitis is brought about is by infective thrombosis of a venule, say of the tympanic roof, continuous with a meningeal vein.

4. *Tuberculosis.*—Tuberculous infection of the temporal bone is by no means uncommon in children, and may be the starting-point of other similar lesions elsewhere. At autopsies it is sometimes difficult to determine which among many tuberculous foci was the primary. The secondary lesions of tuberculous temporal bone disease are commonly in the glands of the neck, in the cerebral meninges, and in the lungs. The importance, therefore, of complete removal of such primary disease is obvious, and in some cases the operation may arrest a secondary meningeal infection. Untreated tuberculous disease in this region may cause sudden and even fatal haemorrhage from ulceration of the internal carotid artery. Tuberculous meningitis, tuberculous abscess, and tuberculous tumour may arise by infection extending directly from the temporal bone, but these lesions are fully as frequently, or even more often, the result of infection carried from a distance by the blood-stream.

Symptoms.—The symptoms met with in the intracranial complications of temporal bone disease are complex and varied: some are referable to the lesion in the temporal bone itself, others to venous infection, some to increased intracranial tension, some to disease of a particular part of the brain, and some to general infection. Some symptoms are more or less common to the group at large, others exist as definite marks of distinction of each several species. Subtle and refined as these may be, they are still within the view of those who by experience and observation have become familiar with such cases. In some cases a diagnosis is easily made, in others with great difficulty only, and when more than one intracranial complication is present the sagacity of the practitioner may be severely tried. However insidiously these cases may arise they generally come for treatment as acute cases, which demand prompt surgical intervention. The causative ear-disease is most often chronic, especially in the sinus infections, and its presence is generally, though not always, obvious. The clinical history shews that for years there has been continuous or intermittent purulent discharge from the ear, which often ceases a little before the onset of the acute symptoms. On inspection perforation of the tympanic membrane is seen, with pus in the meatus, often offensive, granulation-tissue, and occasionally fragments of cholesteatoma. Bare bone can sometimes be felt with a probe, and very offensive fragments are sometimes washed away by gentle syringing. Sometimes distinct bulging of the posterior wall of the meatus or a definite carious track is observed. In a few cases, however, and these are particularly dangerous, the aural discharge has only appeared occasionally, and has never been abundant; the tympanic perforation is small, and may even be closed at the time of examination. In such cases no history of the ear disease may be obtained. Moreover, it is quite possible, especially in young children, for the disease to reach the brain before the tympanic membrane is perforated. The arrangement of the mucous pouches in the upper part of the tympanum tends to confine pus to this region. The incomplete ossification of the tegmen and the free venous communications in early life facilitate intracranial infection. In tuberculosis the discharge is often thin and watery, and may escape observation, or be considered unimportant, so that tuberculous glands may be removed from the neck while the primary disease in the ear may remain unnoticed and untouched.

It will be convenient to discuss the symptoms under the following headings: (1) meningitis serosa; (2) meningitis suppurativa; (3) meningitis tuberculosa; (4) abscess of the brain, including extradural abscess; (5) venous infection.

1. By *meningitis serosa* is meant an accumulation of fluid, as an acute or subacute condition, in the subdural space, the subarachnoid space, and in the cerebral ventricles. The intracranial serous membranes, just like the others, are excited to hypersecretion by toxins brought to them by the circulating blood, as in fevers, or by their falling within the sphere of toxic influence exerted directly by an infective focus upon the immediately surrounding area. It is well known that an infective focus in the neighbourhood of a serous membrane may, without visible or demonstrable direct extension to the membrane, so influence it as to lead to a considerable serous effusion into the cavity; for instance, an abundant pleural effusion, which for a long time may remain serous, may be due to caries of rib or to an abscess deeply placed between the lobes of a lung and quite shut off from the general pleural cavity. In the old operation for opening a pyosalpinx from the vagina a serous effusion is commonly first met with, and only after penetrating farther is the purulent pouch opened. In like manner an extradural suppurative focus may, and sometimes does, give rise to a subdural serous effusion. In the skull the conditions seldom admit of so clear an anatomical demonstration as in the pleura and in the pelvis, and so the evidence of the occurrence of meningitis serosa ex otitide is clinical rather than anatomical, but there is no doubt that it is the lesion causing certain clinical phenomena, although it should be remembered that a very small and easily overlooked lesion may in the meninges, as in the subcutaneous tissue, set up a considerable amount of surrounding oedema.

The serotoxic inflammation is ordinarily diffuse, but does not necessarily extend equally over the whole surface of the brain; it may be more or less completely restricted to the particular portion of the brain nearest to the focus of suppuration, which is the source of the toxin. In middle-ear disease the part of the brain so affected is either the cerebellum or the temporo-sphenoidal lobe of the cerebrum. The inflammation not unfrequently spreads to the cerebral cortex underlying the affected meninges, the pathological condition present being a serotoxic meningo-encephalitis rather than simply meningitis serosa. The symptoms are those of increased intracranial tension and cerebral irritation, sometimes accompanied by more or less decided symptoms of disease of some particular portion of the brain. Patients do not die in the stage of localised cerebral oedema, but the symptoms present may so strongly suggest the presence of abscess as to lead to incision of the brain. "In none of the cases really or apparently cured by operation was the condition correctly diagnosed before the intracranial operation: it was never the course but always the subsidence of the disease which led to the case being included in the category of meningo-encephalitis serosa." Körner also points out that serous meningitis is most frequent during youth or early adult life, 75 per cent of the cases occurring in patients under twenty-five years of age, and 45 per cent between the tenth and the fifteenth year. In certain cases of acute mastoid inflammation some portion of dura exposed at the operation may be found to be pink in colour. This inflammation of the dura is often associated with symptoms of meningitis serosa, such as headache, drowsiness, rise of temperature, relatively slow pulse, restlessness, vomiting, and dilated sluggish pupils. Lumbar puncture gives immediate and striking relief in most of these cases, and may prevent a serous meningitis from becoming purulent.

2. *Meningitis Suppurativa.*—There is no one pathognomonic symptom of meningitis. The symptoms which arise are not the direct result of the meningeal lesion, but are largely due to the influence exercised by the inflamed meninges on the brain substance beneath. Until quite recently diagnosis depended upon symptoms alone, but within recent years lumbar puncture has provided a valuable though indirect means of physical examination. Though most of, if not all, the symptoms met with in cases of meningitis are also seen in other conditions, yet clinical experience has taught us that a particular grouping of certain symptoms is usually associated with manifest meningeal lesions. In seeking to define the relation of symptoms to lesions and to apportion to each symptom its exact diagnostic significance, we are met by the initial difficulty that, on the one hand, the symptoms are sometimes present without demonstrable meningeal lesion, and on the other hand, that autopsies sometimes reveal gross meningeal changes which had been quite unsuspected during life.

Our present knowledge seems to shew that the symptoms most directly referable to the meningeal inflammation are headache, vomiting, constipation, and squint or other evidence of implication of the cranial nerves; these are the cardinal symptoms of meningitis. The headache is severe and persistent, the vomiting apparently purposeless and not accompanied by nausea, and the constipation is obstinate, resisting purgatives, and neither accompanied by abdominal distension nor associated with abdominal pain. The symptoms appear to depend mainly upon intracranial effusion, whereby the pressure relations are altered and the normal power of adjustment of the intracranial tension impaired, but in some degree also upon absorption of toxins. Such fibrous structures as the dura, the sclerotic, and the aponeuroses are, in certain circumstances, highly sensitive, incision of them is distinctly painful, and severe pain is caused when they are subjected to acute tension. The headache of meningitis is comparable to the eye-ache of glaucoma; both are due to tension of a fibrous envelope enclosing a nervous tissue.

With these cardinal symptoms are associated two other groups of symptoms: (*A*) symptoms, such as fever and impaired nutrition, resulting from general infection, and depending more upon the form of the infection than upon the distribution or degree of the meningeal lesions; (*B*) symptoms which are the clinical expression, not of the meningeal lesions, but of the irritation of the subjacent cortex. These vary with the nature, degree, and distribution of the meningeal lesions, and with the cortical irritability of the individual. Most of the symptoms met with in cases of meningitis belong to this group. The mental condition may be altered, the patient, especially if a child, becoming fretful and irritable and frequently screaming. There may be various motor disturbances, most prominent among which are convulsions, retraction of head and the phenomenon known as Kernig's sign. There may be photophobia and more or less general hyperaesthesia. There may be vasomotor disturbances, of which the well-known *tache cérébrale* is an example. In the later stages paralysis, anaesthesia, and coma occur from exhaustion and death of nerve cells. We should expect to find optic neuritis in most cases of meningitis, and it may be an important early diagnostic sign. Körner says, "Curiously enough optic neuritis is almost invariably absent in uncomplicated cases." The examination of the fundus in young children frequently presents difficulties unless the patient is anaesthetised, and wild maniacal delirium is frequent in suppurative meningitis in the adult. Not unfrequently symptoms of extension of the inflammation to the spinal meninges occur, such as pain in the sacral region or about the anus, or incontinence of urine and faeces. Brieger has described a peculiar form of meningitis running an intermittent course. At the seat of infection there is a circumscribed focus of meningeal inflammation; this often determines symptoms lasting a considerable time, but so slight that they are scarcely noticed, or at all events not rightly interpreted; but at intervals this focus sets up an inflammatory oedema, spreading through the arachnoid and causing increase in the cerebrospinal fluid, in fact bringing about the condition known as meningitis serosa. This may subside either spontaneously or after an operation, whereby the disease again enters into a phase of more or less complete latency, until at last the localised meningeal inflammation is either definitively removed by operation or leads to general suppurative meningitis.

3. *Tuberculous meningitis* is variously associated with ear disease, sometimes the meninges have become directly infected with tubercle from a tuberculous temporal bone, sometimes tuberculous meningitis occurs in the course of chronic suppuration in the temporal bone, and

sometimes aural suppuration occurs late in tuberculosis. Suppurative tuberculous disease of the temporal bone by the time it comes under treatment is always a mixed infection, and from such a bone the meninges may be infected with non-tuberculous suppuration and not necessarily with tubercle, so that ordinary suppurative meningitis may occur as a complication of tuberculous bone disease. In the child operation on tuberculous disease of the temporal and other bones is sometimes followed by death in a short time, say in thirty-six hours, the patient never really reviving from the operation. At the autopsy the meninges shew miliary tubercles which had never given rise to symptoms during life. In other cases the operation is followed, in say three weeks, by symptoms of meningeal tuberculosis, which appears to have been lighted up by the operation. The miliary tuberculosis of very young infants is often very acute and rapid, death occurring in two or three days.

Though the symptoms of tuberculous meningitis present considerable variation in individual cases, and there is no single symptom so readily recognised and so constant that it can be regarded as a diagnostic sign, the disease can generally be recognised clinically from the course of the case, the manner of grouping of the symptoms, and the relative prominence of certain signs. The onset of tuberculous meningitis is commonly insidious, and is often preceded by prodromal symptoms such as emaciation and general ill-health, and the course of the disease is slow. Often, but by no means always, it is more or less clearly divided into the three stages so well described many years ago by Robert Whytt. Spinal phenomena are less frequent and less pronounced than in suppurative meningitis; there is very frequently at an early stage squint, inequality of pupils or other sign of affection of the third nerve, and cerebral symptoms are generally prominent. Not unfrequently there are focal cerebral symptoms, sometimes explained by localised patches of tubercle, sometimes without demonstrable anatomical lesion. Such focal symptoms are not the same throughout the course of the case, but may vary from day to day. Constipation is generally obstinate, the abdominal wall becomes retracted, sometimes to an extreme degree. The pulse is commonly quick and irregular. Optic neuritis is not always present in the early stage, though it usually occurs during the course of the disease, sometimes tubercles in the choroid can be seen, but they are only occasionally to be detected and may exist apart from meningitis. The tubercle bacillus can sometimes be detected in the cerebrospinal fluid obtained by lumbar puncture.

4. *Extradural Abscess and Brain Abscess.*—The bone disease giving rise to brain abscess is not characterised by any distinctive feature, but it is sometimes noticeable that after cleansing the meatus and tympanum the whole passage immediately refills with pus; this suggests that the pus comes from a large cavity, and is almost characteristic of abscess beyond the limits of the temporal bone, though it does not in any way distinguish between extra- and intra-dural abscess. It is an important

sign, for when pus is thus able to escape through the tympanum and meatus the distinctive symptoms of brain abscess may be slight and undecided, or altogether absent owing to the relief of tension. Suppuration between the bone and dura gives rise to no specific symptoms, and the first indication of the presence of an extradural abscess is often the discovery of the pus during the course of an operation for disease of the bone. When the pus happens to be under tension there is much local pain and fever, possibly there may be tenderness on percussion over the site of the abscess, and when the suppuration is in the posterior fossa there is often rigidity of the neck. Sometimes symptoms arise from compression of the brain, but there is then nothing to distinguish extradural from intradural suppuration. When the infection is virulent enough to make its way rapidly through the dura, the pus not being under tension, the extradural stage of the progress of the case is but seldom attended by any recognisable symptoms. Extradural abscess is most frequent in the posterior fossa. Infection sometimes reaches the dura, not by direct contact with diseased bone, but along a small meningeal vessel passing through the diseased bone; in such cases the tympanum may heal and the extradural abscess continue to increase or perforate the dura. I have seen this in several cases of influenzal otitis.

We may classify the symptoms of brain abscess as: (i.) those due to the mere presence anywhere in the body of deep-seated pus, such as the febrile state with, perhaps, shivering and vomiting; (ii.) those due to increase of tension within the closed cavity of the skull, such as purposeless vomiting, slow pulse, torpor; (iii.) those due to irritation or suppression of function of particular parts of the central nervous system, such as epilepsy, anaesthesia, paralysis, and perversion or loss of one or other of the special senses. The symptoms of brain abscess are sometimes pathognomonic as to its situation; in others they are in this respect indefinite, and the diagnosis of the seat of the abscess, if possible at all, must be made from the attending circumstances rather than from the direct effects of the abscess on the brain. The symptoms common to all cases of abscess of brain are headache, vomiting, vertigo, photophobia, slow cerebration, drowsiness, optic neuritis, low temperature, slow pulse with irregular rhythm, slow respiration, foul breath, constipation, emaciation, pallor of the face, and expressionless countenance. Any of these, or all, may be present some time or other during the course of an individual case, but none affords definite information as to the particular part of the brain affected. The causative ear-disease is usually of long standing, and very often there is a history of sudden onset of illness with earache, headache, fever, and sometimes shivering; these symptoms, however, do not indicate the commencement of brain infection, but a stage in the evolution of the abscess which, unless surgical relief be afforded, will soon end in the death of the patient.

Nothing is easier than to diagnose the site of a gross lesion when certain well-characterised clinical manifestations are present, but in many instances such pathognomonic signs are absent, or can only be detected by repeated careful examination. The pathological process is ingravescent, and what may be wanting when looked for to-day may be present to-morrow. The patient's condition may, however, demand an immediate operation, and there may be some symptom, such as persistent local pain or incessant vomiting, which, though not characteristic, is yet so much in favour of a particular localisation as to determine the site of operation. In some cases the direction which the disease is taking may be ascertained by careful examination with speculum and probe, or becomes clear during the course of the bone operation. To await the complete evolution of all the possible symptoms of a particular brain abscess before operating would be like waiting for the complete evolution of acute appendicitis through all its stages, including general peritonitis and the death of the patient.

Otitic brain abscess is almost always in the cerebellum or in the temporo-sphenoidal lobe; other original localisations or extensions occur, but they are rare. My own experience is that abscess of the cerebellum is more frequent than temporo-sphenoidal abscess, but Körner has come to the opposite conclusion. The localising symptoms of abscess of the temporo-sphenoidal lobe result from disturbance of cortical centres, or from pressure on nerves, or extensions of the inflammation to adjacent parts of the brain. In the temporo-sphenoidal lobe are the cortical centres for hearing and for taste and smell as demonstrated by Dr. Ferrier's well-known experiments, and often verified by clinical observation. When the abscess is in the apex of the lobe hallucinations or loss of smell may be met with. When the cortical centre for hearing is affected there will be hyperacusia, tinnitus, or hallucinations of hearing if the lesion be irritative, or, if the lesion be destructive, deafness referred to the ear of the opposite side. The cortical centres for the mechanism of language are, in right-handed individuals, on the left side of the brain. The motor speech-centre of Broca, the auditory word-centre, and the visual word-centre, are those likely to be affected in temporo-sphenoidal abscess, and hence various degrees of aphasia are met with as symptoms of a left-sided abscess in that region. In several cases the speech defect has been limited to inability to correlate the ideas of objects with their names. A temporo-sphenoidal abscess on the left side is, because of the speech defects that arise, commonly more easy to recognise than one on the right. The abscess may extend so as to disturb the cortical motor and sensory centres for the face and limbs, or to the internal capsule, and so give rise to contra-lateral hemiplegia, either (*a*) of cortical origin —the face being first affected, then the arm, and lastly the leg; or (*b*) of internal capsule type, the paralysis spreading in the order—leg, arm, face. The sensory defects of cortical origin are partial hemianaesthesia on the contra-lateral side with loss of sense of position, atopognosis, and astereognosis; they are most pronounced in the limb corresponding to the cortical centre chiefly affected. Pressure on the sensory radiation at the back of the internal capsule causes complete hemianaesthesia of the opposite side for all forms of sensation. Occasionally, convulsions of the face and limbs, beginning on the contra-lateral side, are observed. The abscess frequently causes pressure on, and more or less complete paralysis of, the third nerve on the same side: a stabile pupil on the side of the lesion is very characteristic of temporo-sphenoidal abscess, and often clinches the diagnosis. A peculiar mental condition, known as the "dreamy state," in many cases results from temporo-sphenoidal abscess on either side. I have observed this phenomenon more particularly in cases of right-sided temporo-sphenoidal abscess, probably because of the absence of other symptoms. The deep reflexes are commonly exaggerated on the contra-lateral side.

The symptoms characteristic of a cerebellar lesion are vertigo and certain disturbances of equilibrium and movement. The vertigo occurs early, is often severe, is intensified by change of position, and is associated with a feeling of weakness and faintness. Vertigo alone is not proof of cerebellar disease, as it is often due to disease limited to the labyrinth. The patient stands with the legs widely separated, the abduction being greater on the side of the lesion; in walking he takes a zigzag instead of a straight course, the greater deviation from the straight line being towards the side of the lesion. The patient is unable to turn sharply at the word of command. There is often a tendency to fall towards the side of the lesion. Romberg's sign may or may not be present. There is ataxy of the homo-lateral limbs, but it is sometimes present on both sides, particularly when the vermis is affected; weakness and loss of muscular tone in the homolateral limbs, most decided in the arm, and weakness of both lower limbs. Occasionally we find rigidity and spasm of the limbs, fixed attitude of head, forced position in bed, movements of rotation, nystagmus—especially horizontal nystagmus—when the patient looks away from the side of the lesion, conjugate deviation of the eyes to the opposite side, or skew deviation of the eyes. The deep reflexes may be exaggerated or diminished, but any alteration is on the homo-lateral side. Characteristic cerebellar symptoms are most pronounced in lesions of the vermis, a region not often primarily the site of abscess, but sometimes secondarily affected by pressure, or by extension of the abscess or inflammation; they do not occur in small lesions of the lateral lobe situated external to, and not causing pressure on, the intra-cerebellar paths of the upper and lower cerebellar peduncles, and of the nucleus dentatus. Many otitic cerebellar abscesses are so placed. There is no cutaneous anaesthesia, and deafness, when present, is on the side of the lesion, and due to the bone disease, or to compression or destruction of the auditory nerve. Some peculiarities of the general symptoms of intracranial lesion, though by no means characteristic, yet strongly suggest that the site of the abscess is in the cerebellum; thus, optic neuritis is commonly early and severe, and more intense on the side of the lesion; vomiting is obstinate and frequent, and there is often rapid emaciation. Sometimes the head is retracted from the irritation of concomitant posterior basal meningitis.

The clinical course of a brain abscess can be more or less sharply divided into four stages—the initial, the latent, the manifest, and the terminal. The initial stage, accompanied by fever, headache, and vomiting, is usually present, but of short duration, and, as it presents nothing characteristic, its significance is often not appreciated. The latency of the next stage is not often absolute, though the symptoms present must be carefully sought for; occasionally, however, the patient is apparently quite well until a few days before death.

5. *Venous Infection.*—When the condition is well established, the symptoms are those of septicaemia or pyaemia, often, but not always, preceded or accompanied by symptoms referable to the bone disease, or to infection and thrombosis of a particular sinus. Septicaemia is characterised by high fever with remissions, occasionally with rigors in the initial stage, but seldom with a succession of rigors; pyaemia by high fever with remissions of several degrees, sweating, frequent rigors, and metastatic abscesses. Septicaemia and pyaemia are not separate and distinct diseases, but phases of the septic process. Vomiting frequently occurs as the infection first becomes generalised, but is not commonly persistent or frequent. It is the same in other forms of septic infection, notably in puerperal septicaemia, of which vomiting and shivering together constitute an early and dangerous indication. It is misleading to speak of such symptoms as symptoms of lateral sinus thrombosis, for they are not symptoms of thrombosis, but of general infection. The danger in these cases is not thrombosis, but infection of the thrombus and generalisation of the infection. Thrombosis is really a protective measure adopted by nature to limit the spread of infection, and may be completely successful.

In the early stage of venous infection there is often severe pain in the affected ear with agonising headache, and sometimes there is meningitis serosa. Ophthalmoscopic examination often shews extreme venous congestion of the disc without definite inflammatory swelling. When, as is usually the case, one of the larger sinuses is affected, certain symptoms due to obstruction and inflammation of the particular sinus are frequently, but not invariably, present. In sigmoid sinus thrombosis, by far the most frequently met with, such manifestations are oedema and tenderness over the mastoid, particularly along its posterior border; tenderness along the internal jugular vein, and enlarged and tender cervical glands. Sometimes a thrombus can be felt in the internal jugular vein. The vagus, the spinal accessory, and the glossopharyngeal nerves at the foramen lacerum posticum may participate in the inflammation, and, when the infection extends backwards along the anterior condyloid vein, the hypoglossal at the anterior condyloid foramen may likewise be affected; as a result, the pulse may become intermittent, there may be cough and disturbance of the respiratory rhythm, or painful contraction of the sterno-mastoid and trapezius muscles, difficulty in swallowing, and spasm or paresis of the muscles supplied by the hypoglossal nerve.

Thrombosis of the cavernous sinus gives rise to oedema of the eyelids and orbits, exophthalmos, retinal congestion and choked disc, neuralgia of the first division of the fifth nerve, and more or less complete paralysis of the muscles supplied by the third, fourth, and sixth nerves. In thrombosis of the superior longitudinal sinus there is sometimes oedema of the scalp in the neighbourhood of the sagittal suture. The local effects of thrombosis of the other sinuses are confined to the interior of the skull, and consequently not recognisable externally.

Some further Remarks on Symptoms and Diagnosis.—In many cases the symptoms of one or other of the intracranial complications are fairly complete and uncomplicated, but by no means unfrequently the surgeon has to act when the symptoms are less characteristic, or not confined to those ordinarily associated with one particular morbid condition. Brain abscess presents peculiar difficulties when it runs a latent course and when it is complicated with other conditions. The lack of definite localising symptoms is sometimes most striking, especially in cerebellar abscess, and the opportunity for successful operation is often lost from the condition of the brain escaping recognition or localisation. One of the most important signs of an extending brain abscess is vomiting, though in some cases of temporo-sphenoidal abscess it is not prominent. The difficulty of diagnosis is often increased by the patient not being accurately examined until the intelligence is so much impaired that a complete examination is impossible. The history of ear trouble is often unknown to the friends of the patient, especially in hospital practice; and their story, too, of the days of illness previous to admission is for the most part clinically valueless. In some cases of chronic otorrhoea there is optic neuritis without any other symptom of intracranial mischief, and the question then arises whether any other complication, such as latent brain abscess, is present. I have observed several such cases. The optic neuritis is slight, and may last for several months without impairing sight. It subsides when the temporal bone disease is efficiently removed. Disease of so many parts of the brain disturbs the innervation of the ocular muscles that nystagmus is only of value in the diagnosis of cerebellar abscess when associated with other signs. Horizontal nystagmus is associated with disease of the lateral lobe of the cerebellum : the larger excursions of the eyeballs are observed when the eyes are directed towards the side of the lesion ; nystagmus in all directions occurs in disease of the middle lobe. The symptoms of cerebellar abscess are often masked by those due to distension of the ventricles. Abscess in the cerebellum and in the temporo-sphenoidal lobe, like abscess elsewhere in the brain, may be latent, producing only general symptoms of ill-health, until excited to renewed activity by a febrile attack, by a blow on the head, or by some minor operation such as the removal of a polypus. A patient with ear disease may have an abscess in any part of the brain from some other cause, such as influenza. A voracious appetite is frequently noticed in convalescence from brain abscess, and is one of the best indications of satisfactory progress.

Abscess may occur simultaneously in more than one part of the brain, for example, in the cerebellum and in the temporo-sphenoidal lobe, and exact diagnosis would be difficult, if not impossible, but it has happened in practice that both abscesses have been opened. When meningitis complicates a brain abscess the symptoms due to the brain abscess will be modified or controlled by those due to the meningitis, the temperature is relatively high, the pulse is quick, delirium, convulsions, and optic neuritis occur early, pain in the head is severe, the head may be retracted, and vomiting, squint, and irregular respiration may be prominent symptoms. Brain abscess, especially cerebellar abscess, is often associated with sinus pyaemia, in most instances the symptoms of sinus infection precede those of the brain abscess. As the abscess increases the mental state becomes impaired, and the lower temperature and slower pulse of abscess take the place of the oscillating temperature and rapid pulse of pyaemia. Acute hydrocephalus is no uncommon complication of cerebellar abscess. If an abscess burst or leak into one of the ventricles general purulent infection of the ependyma occurs; this is quickly fatal, usually with coma and high fever.

Two modern methods of diagnosis often furnish valuable assistance in the diagnosis of the morbid conditions under consideration: (1) The microscopic examination of the blood. Leucocytosis of the blood in these as in other suppurations is, when observed, a valuable aid in diagnosis; I have recently seen marked leucocytosis of the blood in a case of large cerebral abscess, and on the other hand in another case of cerebral abscess, which was freely discharging externally, there was no increase in the number of white blood cells. Sonnenburg, writing of suppuration in another region of the body, says that leucocytosis of the blood is the expression of the reaction of the organism to infection, and adds the important statement that when the leucocytosis is high, that is 20,000 or over per c.mm., the prognosis of operation is favourable notwithstanding the severity of other symptoms, but that a low or absent leucocytosis with severe symptoms is an absolutely bad sign. (2) The operation of lumbar puncture enables us to estimate the intradural pressure, and by examining the fluid withdrawn we are able to obtain information on various points of diagnostic significance; the most important points in the present connexion are (*a*) The cells. The general indications are that a mononuclear leucocytosis points to a slow or subsiding inflammation, and abundance of polymorphonuclears to an active and intense inflammation. The presence of cells in the fluid withdrawn by lumbar puncture is indicative of the presence of meningitis. In a recent fatal case of basal meningitis the fluid was visibly turbid with cells, the majority of which were polymorphonuclear. (*b*) The bacteriology. Tubercle bacilli are demonstrable, particularly in the lymph flakes that form, in a large proportion of, but not in all, tuberculous cases. The *Meningococcus*, the *Pneumococcus*, the *Bacillus coli communis*, streptococci, and staphylococci have also been found as the causative organism. (*c*) The amount of albumin. In normal fluid there is none, or the merest trace; heavy traces of albumin indicate severe inflammation. Dr. Graham Forbes has recently published a valuable paper shewing the results obtained by examination of 140 specimens of cerebrospinal fluid.

The symptoms of some other diseases bear a certain resemblance to those of abscess of the brain. *Tuberculous meningitis* in children is often associated with purulent otorrhoea, and certain localising symptoms, particularly hemiplegia, sometimes predominate and lead to a suspicion of brain abscess. The salient features in which such a case of tuberculous meningitis differs from one of brain abscess are: the temperature is above normal, the pulse is 100 or more rapid, and the child is dull or irritable even before illness is suspected.

Marantic thrombosis of the cerebral sinuses is sometimes associated with ear disease and paralysis. The symptoms differ from those of brain abscess in that the temperature and the rate of the pulse are above the normal. The degree of ear disease is slight, and the paralyses, especially of the face, are not constant but alternating. Marantic thrombosis is a disease almost exclusively of childhood; but cerebral embolism, thrombosis, and haemorrhage in the adult may, when ear disease happens to be present, cause some embarrassment in diagnosis. In abscess of the brain the onset of the symptoms is gradual, being spread over days or weeks; in embolism and haemorrhage the onset is instantaneous, whilst in thrombosis the manifestation of the condition may extend over hours or days; and morbid conditions, such as chronic cardiac or renal disease, which usually accompany these vascular lesions, may be detected.

Encephalitis Non-suppurativa.—In their recent work on encephalitis Oppenheim and Cassirer point out that the brain, like other parts of the body, is subject to non-suppurative inflammation. They distinguish certain groups of cases—(i.) poliencephalitis haemorrhagica superior in which ophthalmoplegia externa is the most prominent symptom; (ii.) poliencephalitis inferior, in which acute bulbar paralysis forms the central symptom; (iii.) poliencephalo-myelitis, in which spinal as well as cerebral nerve nuclei are affected; (iv.) acute primary haemorrhagic encephalitis, a disease of an infective character affecting mainly the cerebral hemispheres and occurring most frequently in connexion with influenza. In this last group of cases the diagnosis from brain abscess, meningitis, and infective sinus thrombosis sometimes presents considerable difficulties, indeed in one case an operation was needlessly performed on the temporal bone and sinus transversus, the patient's disease being really this form of encephalitis. Symptoms resembling or even to all appearance quite identical with those of the various forms of encephalitis are sometimes met with in cases in which the most careful anatomical examination has failed to detect any demonstrable lesion; and sometimes patients presenting characteristic symptoms of encephalitis recover rapidly and completely, from which it would be inferred that no gross morbid change has taken place.

Principles of Treatment.—The ideal treatment of all the intracranial complications of ear disease is the preventive. Chronic persistent infective disease of the temporal bone should be treated by an appropriate operation before it reaches the interior of the skull, and acute temporal bone infections should be treated surgically like acute infections of other bones. In this way the intracranial complications which may follow any case of temporal bone disease, either in its acute stage or long years after its commencement, would for the most part be obviated. In this article the indications for operation apart from threatened or actual intracranial complications are not discussed. In all the intracranial complications the only treatment is operation, the nature and scope of which necessarily vary with the particular complication present. The earlier operation is undertaken the greater the chance of success; the fate of most of these cases lies in the hands of the general practitioner, who should be alive to their gravity, and lose no time in seeking such aid as may be necessary for dealing with them. In some cases the surgeon can come to a decision before operating as to the exact procedure required; in others he must be guided by what he finds in the course of the operation. It is important to remember that very severe general symptoms, with vertigo, vomiting, and optic neuritis, may be caused by retention of pus within the temporal bone, and complete recovery may then take place after an operation limited to the bone; on the other hand, when the symptoms are conclusive of the presence of pus beyond the limits of the temporal bone, it would be futile to be content with the bone operation alone whatever the disease found within it. The first step is always complete exposure of the disease in the bone, which in almost all cases has commenced in the tympano-antral cavity.

There are two types of dangerous cases of disease in the temporal bone, one in which a definite track of disease leads from the temporal bone into the interior of the skull, giving rise to meningitis, brain abscess, or sinus infection, and another in which a blood infection of sapraemic, septicaemic, or pyaemic character arises from infection of the small veins in the bone itself, without any thrombosis of the larger sinuses. Spread of infection to the interior of the skull by continuity is usually the result of chronic disease. Disease may spread by continuity from the temporal bone to the interior of the skull either by making a new track for itself throughout, or by first opening the labyrinth by penetrating one of the fenestrae or the bony wall of the external semicircular canal, and then traversing one or other of the anatomical channels of the labyrinth, appearing on the intracranial surface at the internal auditory meatus or at the saccus endolymphaticus. A knowledge of the path taken by the disease determines the operative measures to be adopted, for the rule of treatment is that the disease must be followed through the channel by which it has traversed the temporal bone. The measures adopted to each particular complication will now be considered.

Extradural Abscess.—Pus is not uncommonly found external to the dura when nothing in the symptoms has led the surgeon to suspect it. In some cases such an abscess is opened up in following a carious track through the bone, in others by removing the tegmen or the bony boundary of the posterior fossa though not visibly perforated by the disease. Infection so frequently proceeds through the tegmen by an aperture not sufficiently large to attract attention that I recommend the removal of the tegmen, whether obviously carious or not, in all cases in which there has been reason to suspect intracranial complication, and indeed almost as a routine measure in the complete mastoid operation. There are few, if any, cases in which an operation undertaken for suspected intracranial complication should not be carried down to the dura. The dura may appear healthy, may be granulating, may have sloughed, or there may be a more or less considerable extradural abscess. Necrotic dura should be excised, healthy or granulating dura should be left alone unless symptoms have been observed which necessitate an intradural exploration. Sufficient bone should be removed to open up the whole cavity of any extradural abscess or to expose the whole of any granulations on the dura.

Brain Abscess.—The object of the surgeon is to obtain complete evacuation and drainage of the abscess. Whenever possible it should be opened and drained through its "stalk"; for in this way the patient runs no risk either of cerebral hernia or of meningitis. When, for any reason, it is desirable to open the abscess at a spot where the meninges are not adherent, either at the primary operation or to establish a counter-opening, it is advisable first to expose the brain, and then to wait until the meninges have become adherent before incising it, on the principle that used to be adopted in some abdominal operations. It is often impossible to follow this ideal plan, because the desperate condition of the patient precludes delay. The subdural space can to some extent be protected, like the peritoneum, by gauze packing during an operation, but by no means so efficiently, for the brain cannot be handled with the same facility as the abdominal viscera, and though it bulges against the opening into the skull before the abscess is incised, it will collapse after the evacuation of the pus. Packing the subdural space, moreover, does not protect the subarachnoid space from infection. It is advisable to introduce more than one drainage-tube into the cavity of a brain abscess, and, when once in place, the tube, or tubes, should not be removed until no longer likely to be needed, since, the brain being of liquid texture, the boundaries of the abscess cavity are apt to flow together, and thus prevent proper reintroduction. At the end of a week one tube may be removed, and, unless the abscess cavity be very large, the other at the end of a fortnight. Irrigation of the abscess cavity is not advisable. The dressing should be changed daily, the external wound should be washed with diluted chlorinated soda solution (one part of liquor sodae chlorinatae, B.P., in 20), to get rid of any discharge that may have escaped and to prevent decomposition on the surface of the wound. In operating for temporo-sphenoidal abscess the stalk of the abscess should be looked for, adherent to the upper surface of the tegmen; in cerebellar abscess the stalk will commonly be found adherent to some portion of the dura over the posterior surface of the petrous internal to the sigmoid sinus. It has often happened in cases of cerebellar abscess that respiration has ceased during the first few inhalations of chloroform; this is not a bar to the successful conclusion of the operation, but is an urgent reason for completing it while respiration is maintained artificially.

After the drainage of a brain abscess, whatever the symptoms of further cerebral trouble that may arise, and they often seem inexplicable, the brain should be explored through the original wound, and no chance operation undertaken in another region. Instead of concentrating the attention on the original site of abscess, the new symptoms may suggest conditions which are not present, such as meningitis or acute distension of the ventricles; the symptoms being really due to the refilling of the old abscess or to the formation of a new one in the immediate vicinity. The temperature, pulse, and respiration in brain abscess are modified when the dura is opened, in consequence of the relief of intracranial pressure.

Meningitis.—The surgeon may have to deal with serous or suppurative meningitis; either variety may be tuberculous or non-tuberculous. Some cases of meningitis serosa recover after the bone operation alone or combined with lumbar puncture, so that incision of the dura is unnecessary. Other cases recover after incision of the dura, made in the expectation of finding pus. Suppurative meningitis may be localised or diffused. Pus may be found in the subdural space spread out in a sheet of greater or less thickness over a certain limited area. This condition, when occurring as a complication of ear disease, is almost always fatal. Localised suppurative meningitis affecting the subarachnoid space may cover a wide area or be confined to a quite narrow track. The treatment is removal of all bone disease, with provision for free drainage of the affected meninges. In general suppurative meningitis the operation affording the best chance of success is one which provides a free bilateral opening, and allows the pus to escape from the subarachnoid space. Hinsberg has published an instructive paper on this subject. It has been suggested that the spinal theca should also be opened in the lumbar region, as purulent material is early carried down the spinal meninges. Körner says:—"Until quite recently the progress of otitic suppurative meningitis was absolutely lethal. Even at the present time no case of streptococcic meningitis from ear disease has been known to recover, though a few cases of staphylococcic meningitis have recovered after early operation" (8).

Tuberculous Meningitis.—Cases presenting apparently decided symptoms of this condition have recovered after free removal of the temporal bone disease. If the symptoms persist after the temporal bone operation, it is conceivable that the disease might be arrested by opening up the Sylvian lakes, but so far no successful operation of this nature is on record. Symptoms of meningitis arising for the first time after the bone operation should be treated in the first instance by lumbar puncture. This will not only relieve the symptoms of intracranial pressure, but may arrest the disease, and will in any event afford valuable information as to the fluid with which the meningeal spaces are distended.

Treatment of Venous Infection.—When general infection has taken place through the small veins in the bone itself, and there has been no preliminary infection of the sigmoid or other large sinus, the surgeon, after removal of the bone disease, may decide to depend upon the use of an appropriate antitoxin and upon general remedies. On the other hand, I consider that when the case is seen early an artificial thrombosis should be brought about in the sigmoid sinus, on the grounds that the most direct channel of infection is the internal jugular vein, and that thrombosis of venous trunks is Nature's own way of isolating an infected area from the general circulation. In order to do this the sigmoid sinus should be opened and plugged with gauze, the internal jugular vein divided between two ligatures, and the lower end of the upper segment brought out through the upper end of the skin wound in the neck. The ligature on the segment of vein which has been so brought out should be removed as soon as clotting has taken place. Possibly the method of treatment by terebene injections, which has been found efficacious by Poncet of Lyon in the treatment of puerperal sepsis, might be of use in these cases.

In the more usual variety of venous infection, in which the sigmoid sinus has first been thrombosed, and the thrombus therein become secondarily infected and is extending, the surgeon has not only to deal with the bone disease but with the infected sinus. "Whether the operator should content himself with removing the bone disease and any parasinuous abscess that may be present, depends chiefly upon the general condition of the patient. If there has been no fever the sinus should be let alone, even when covered with granulations or containing a palpable thrombus" (8). When it is certain that infection is spreading by way of the internal jugular vein, the first step in the operation should be ligature of this vessel below the clot. The bone disease should then be removed by the usual operation. The operation on the sigmoid sinus and jugular vein varies greatly in extent. When the condition met with is a local abscess in the sigmoid sinus, isolated at each end by uninfected clot, incision of the abscess suffices for cure. When the clot is infected it should be removed, and the operation must be sufficiently extended to effect this as completely as possible. In extreme cases the vein must be dealt with at its junction with the subclavian, and isolated by ligature of all its tributaries, while the sinus must be exposed as far back as the torcular, and converted into a groove by excision of its external wall. The jugular bulb in some cases requires exposure and incision, but the method advocated by the writer of bringing the upper end of the divided jugular out through the wound in the neck, and taking off the ligature from it, obviates in most cases the necessity for this step. Between the most simple and the most severe case every gradation is met with. The view that operation on the vein is never required, and the view that operation on the vein is always required, are equally erroneous. It is as absurd to tie the vein for a local abscess which should be dealt with as in other parts of the body by simple incision, as it is to decry all operations on the vein when it may contain a spreading infective clot, which has been known to reach the heart.

The cavernous sinus may be primarily and directly infected from the bone disease when this has extended to the apex of the petrous; the infective focus in the sinus is then directly continuous with that in the bone; in other cases infection reaches the cavernous sinus by way of a connecting vein or sinus. Exposure and incision of the sinus afford the only hope of arresting the disease. In the case of Bircher the operator worked his way through the petrous until foul pus escaped from the posterior end of the cavernous sinus. For approaching the cavernous sinus I have adopted the Hartley-Krause method for extirpation of the Gasserian ganglion, and found it easy and effectual, but probably when pus has been evacuated from the sinus, it would be well to adopt the recommendation of Voss, who cuts away the zygoma and removes more bone from the basal aspect of the skull so as to get direct drainage. Operations on the cavernous sinus must be done at an early stage of the infection because of the great facility with which the disease extends to the opposite sinus through the circular and transverse sinuses, and also because the meninges are likely to be infected.

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EUSTACHIAN OBSTRUCTION AND CHRONIC MIDDLE-EAR CATARRH

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EUSTACHIAN obstruction and chronic middle-ear catarrh are intimately related, but it is convenient for practical purposes to consider them under different headings, according as the obstruction of the tube, or the catarrhal symptoms in the tympanum itself, form the more prominent feature.

I. EUSTACHIAN OBSTRUCTION.—Normally the walls of the Eustachian tubes are more or less firmly in contact (8), and are separated by the act of deglutition. When from any cause this separation does not take place a condition of Eustachian obstruction occurs.[1]

Etiology.—The causes of this condition are: (1) slight swelling and stickiness of the Eustachian walls due to catarrh. According to Politzer, "tubal catarrh can rarely be separated clinically from catarrh of the middle ear, and one is justified in speaking of a catarrh of the tube only if by the symptoms the inflammation can be accurately localised in the tube itself without the tympanic cavity being involved in the process" (11). The combined condition is spoken of as tubo-tympanic catarrh. (2) Mechanical interference with the action of the Eustachian muscles (levator and tensor palati) by growths or swellings at the pharyngeal orifice of the tube. (3) Weakness of these muscles, rendering them unable to perform the opening function properly. (4) Organic stricture of the tube, following on chronic catarrh of the middle ear, due to new formation of connective tissue in the submucous layer. This may also occur from cicatrices, syphilis, accidents, and so forth.

Causes 1 and 2, or a combination of the two, are the most common, and come into operation most frequently in children and young adults. Cause 3 is generally considered to be associated with progressive middle-ear deafness, and has been especially studied by Weber-Liel, although his conclusions have scarcely been borne out by subsequent observers.

Organic stricture is comparatively rare, and in my experience cases in which a fine Eustachian bougie cannot be passed up the tube are very infrequent. The seat of the constriction is most often in the cartilaginous portion, but the lumen of the bony portion may be narrowed by hyperostosis of its walls, by bulging of the carotid canal, and at its tympanic extremity, in suppurative cases, by granulations and cicatrices.

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[1] Politzer (9), however, has shewn with the ear manometer (a small curved glass tube containing a drop of coloured fluid, and fitted air-tight into the meatus), that in a number of cases a current of air passes from the pharynx to the tympanic cavity, even during quiet respiration.

Results.—The results of Eustachian obstruction are as follows: the air in the tympanum becomes absorbed, the membrana tympani is driven in by the atmospheric pressure, and varying degrees of loss of hearing and occasionally tinnitus are present.

The symptoms may be deduced from the results just stated. They are: loss of hearing, generally bilateral, retraction of the tympanic membrane, and occasionally tinnitus.

The patient, generally a child, is brought with deafness. The extent of this in a child is best ascertained by testing with the voice. The other ear is closed by an assistant, and the surgeon, standing opposite the ear to be tested, asks easy questions, at first in an ordinary voice, and subsequently in a whisper, the distance at which they are answered being noted. The watch test is, as a rule, quite unreliable in childhood, and the same may be said of the tuning-fork. On examination the membrana tympani is found drawn in (see Fig. 1, Plate XIV.), the handle of the malleus is foreshortened and appears much more horizontal than usual, the small process is very prominent, and the anterior and posterior folds are well marked. The membrane is perhaps somewhat congested, or easily becomes so. On inflation by Politzer's method the hearing is suddenly and completely restored, so that a whispered question is answered across the room. The membrane is found to have regained its normal plane, or, in cases of long standing, it may bulge, like the sail of a ship, both in front of and behind the manubrium, chiefly the latter.

After a varying time, if left alone the deafness recurs, because the catarrhal condition reproduces the Eustachian obstruction. Examination of the nasal cavities from the front shews more or less nasal catarrh, with erection of the inferior turbinated bodies, and after the age of seven years often with more or less deflection of the septum. The tonsils are frequently unhealthy and enlarged. Examination of the naso-pharynx often reveals the presence of adenoid vegetations and post-nasal catarrh. The diagnosis of adenoid vegetations is made either by posterior rhinoscopy, which is often feasible in children if a sufficiently small mirror and cocaine spray to the throat be used, or by palpation. Whenever there is any doubt about the existence of these growths, and indeed usually in order to obtain a clear conception of their size and consistence, palpation should be employed (*vide* p. 89).

The other chief symptom of Eustachian obstruction is tinnitus, which is much more commonly complained of by adults than by children. A sensation of fulness occasionally occurs.

Diagnosis.—The diagnosis rests in children on the *complete* and *immediate* recovery of hearing on successful inflation, on the presence of the indrawn membrane, and in older patients also on the absence of moist sounds in the tympanum when the auscultation tube is used during catheterisation. It is not always easy to ascertain whether moist sounds heard with the auscultation tube on catheterising occur in the Eustachian tube or in the tympanum. In the former case the râles are as a rule more distant and are coarser. In the latter, according to

Dr. M'Bride (5), there is a slight moist sound just preceding the impact of the air upon the membrane. If there be much fluid secretion in the tympanic cavity a very rapid bubble-like rustling and cracking noise is frequently heard, which often lasts for a time after the inflation (10). When there is only a small amount of secretion in the tympanic cavity all gurgling sounds may be absent. When Eustachian obstruction does not yield to inflation by Politzer's method or the catheter, bougies must be employed, and on them the diagnosis must rest in these more obstinate cases of stricture. Any concomitant nerve affection would be indicated by the tuning-fork tests described on p. 351.

The prognosis in ordinary Eustachian obstruction (see p. 496, Cause 1, and also Cause 2) is distinctly favourable. In cases of organic stricture, due to chronic middle-ear catarrh, some improvement in the contraction may be generally obtained with or without a concomitant improvement in hearing power. In cases of cicatrices in the cartilaginous tube the prognosis is very unfavourable.

After-effects.—If neglected, Eustachian obstruction may lead to chronic non-purulent middle-ear conditions of an intractable nature. On this account the treatment of Eustachian obstruction in childhood is of great importance, and is fully deserving of the attention, not only of specialists, but of the general practitioner. Any child who seems stupid or inattentive at school should be carefully tested with a whisper for slight degrees of hardness of hearing, for many a child is punished for inattention whose sole fault is that he is unable to hear perfectly. By thus practising preventive aural surgery it is reasonable to suppose that in the course of time the number of serious cases of chronic aural catarrh, accompanied by intractable changes in the tympanum, amongst the community at large will be appreciably diminished.

The treatment consists in regular inflations of the tympanum. This is done either by Politzer's method (or one of its modifications) or catheterisation, the latter being combined if necessary in severe cases in adults with the passage of a Eustachian bougie. I usually employ celluloid slightly bulbous bougies, and in my experience the distance the bougie should be passed beyond the end of the catheter is usually about two centimetres. Organic strictures should be treated by the passage of bougies in gradually increasing sizes. It is to be noted that inability to pass a bougie does not necessarily prove the presence of a stricture, because the tube may have a congenitally angular form, or the point of the bougie may catch in a fold of the mucous membrane. No force must be used in passing a bougie, or the mucous membrane may be injured and artificial emphysema ensue. Electrolysis and the application of electric currents to the tubes have been recommended in some forms of Eustachian obstruction, but I have not seen any benefit from these forms of treatment.

Having secured the ventilation of the tube, the next step is to prevent a recurrence of the obstruction by the removal of all causes giving rise to it. First of all, adenoids and enlarged tonsils must be removed, the enlarged posterior ends of the inferior turbinated bodies should be removed, and any other cause of nasal obstruction, such as a deflection of the septum, chronic erection or hypertrophy of the anterior ends of the inferior turbinated bodies, appropriately treated. Where none of these exist, or if the catarrh persist after their treatment, the catarrh itself must be treated by sniffing up warm normal saline solution, or a solution of other salts of equal specific gravity, and by the employment of a chloride of ammonium inhaler, the fumes of nascent chloride of ammonium being smoked and blown out of the nose twice a day for ten minutes, and at each sitting being blown twice up the Eustachian tubes by Valsalva's method. A favourite form of astringent spray which I employ for the nose is a mixture of rectified spirit and water, strength of 25 per cent and upwards.

A course of simple exercises to promote nasal respiration should be regularly followed for some months after any obstruction has been removed, and the general treatment for nasal catarrh recommended later on (see p. 502) should be carried out.

As regards adenoids, it may be mentioned that only a moderate amount, insufficient to produce nasal obstruction, may help to keep up catarrh, and on that account require removal in these cases. It is a good plan, as recommended by Mr. Hovell, whenever adenoids are operated upon under an anaesthetic, to pass a snare through each side of the nose and remove any redundant tissue at the posterior ends of the inferior turbinals.

In adults polypi projecting through the choanae not uncommonly give rise to Eustachian obstruction, and in these cases removal of the growths is generally advisable before any treatment of the ears is attempted.

II. CHRONIC CATARRH OF THE MIDDLE EAR may, for practical purposes, be described under two headings: (1) the moist form, accompanied by secretion, either serous or mucous in character; (2) the adhesive form, in which there is no noticeable secretion in the tympanum, but in which there is a tendency to hypertrophy of the tympanic lining and the formation of adhesions between the different intra-tympanic structures. Although it is customary to divide chronic middle-ear catarrh into these two classes, it must be understood that one frequently runs on into the other, and that many cases beginning as catarrhal end in sclerotic changes.

1. **The Moist Form of Chronic Middle-Ear Catarrh.**—(*Otitis Media Catarrhalis Chronica; Otitis Media Serosa.*)

Etiology.—Chronic middle-ear catarrh usually results from repeated attacks of acute tubo-tympanic catarrh, in each of which complete resolution becomes more difficult, until gradually a chronic catarrhal condition of these parts is set up. The cause of these attacks is generally acute nasal or naso-pharyngeal catarrh, which in its turn may be brought about by chills, influenza, acute exanthemata, Bright's disease, syphilis, and so forth. Amongst the causes of chills are undue exposure to cold winds and cold water, as in bathing, and the introduction of cold water into the ear during washing. Middle-ear catarrh is often set up by bacterial invasion from the naso-pharynx, and in children adenoids frequently cause chronic aural catarrh.

Symptoms.—The chief symptoms are loss of hearing, often varying in degree from time to time, tinnitus which, however, is not such a common or pronounced symptom as in adhesive catarrh. The same remark applies to paracusis Willisii (*vide* p. 370). The noises are usually singing, buzzing, occasionally throbbing in character. Pain is scarcely ever present. Occasionally autophony, that is, resonance of the patient's own voice, and giddiness occur.

Examination of the Ear.—There is often an excessive amount of wax in the meatus, and the walls of the canal may be somewhat thickened. The membrana tympani may shew signs of thickening, either localised or general, or it may be atrophied. The thickened areas of the membrane are recognised by being whiter than the normal membrane. There may be calcareous deposits in the membrane, which is often much indrawn with prominence of the anterior and posterior folds. If the membrane be sufficiently thin, fluid mucus in the tympanum may be recognised through it, as will be described under Otitis Media Serosa (p. 502). On inflation, which may be done by Politzer's method, or in adults with a small vulcanite catheter, the membrane bulges more or less, and the manubrial plexus often becomes much injected, in marked contrast to what occurs in the adhesive form of catarrh (see p. 506). On auscultation there are moist râles, which may emanate either from the tube or from the tympanum itself. To distinguish these two practice is needed (see above, p. 497). The tube itself may be narrowed, but conclusions in regard to this point must not be hastily arrived at, as a catheter not placed exactly in the direct axis of the tube may easily give the observer the impression that the tube is narrowed. Inflation produces considerable improvement in hearing, although generally not as much as when the disease is confined to the Eustachian tube. On aspiration with Siegle's speculum the membrane is seen to be more or less fixed, but local atrophied portions may become prominent under this procedure.

Tuning-fork tests indicate that the middle ear alone is affected. With Weber's test the fork is heard better in the worse ear. Except in cases in which the deafness is very slight Rinne's test on the mastoid is negative, especially with low-toned forks. Bone conduction over the mastoid is usually increased, and there is diminished hearing power for low tones.

Examination of the Nose.—The nasal cavities usually shew signs of catarrh, which may be simple, hypertrophic, or associated with growths, such as polypi. There is also catarrh of the naso-pharynx, accompanied often in the young with adenoid growths, and in older persons with hypertrophy of the posterior ends of the inferior turbinated bodies.

Examination of a case of chronic catarrh of the ear should not be considered complete until the nose, and especially the naso-pharynx, have been carefully examined, as to those regions a large and important part of any treatment, to be successful, must be directed. Inspection of the pharynx will also often shew enlargement of the tonsils, thickening of the salpingo-pharyngeal folds, and granular pharyngitis.

The diagnosis of chronic catarrh of the middle ear from adhesive middle-ear catarrh is made: (1) by the evidence of liquid in the tympanum, gained either from examination of the membrane, or more commonly from the intra-tympanic sounds heard on auscultation; (2) by the improvement in hearing produced by inflation of the tympanum. The occurrence of injection of the membrane after inflation also helps to confirm the diagnosis of moist catarrh. The tuning-fork tests, which point to middle-ear disease, are practically the same in the two complaints.

From otosclerosis (capsulitis of the labyrinth) it is distinguished by the normal membrane, through which occasionally a reddish blush is seen, and by the absence of a history of catarrhal symptoms in otosclerosis.

The prognosis in any given case of chronic catarrh of the middle ear depends on the amount of the improvement in the hearing power produced by inflation, and on its duration. Usually the improvement in hearing is considerable, and may last for twenty-four hours or more. The prognosis in chronic catarrh of the middle ear is generally favourable, and by proper treatment considerable improvement can be effected and maintained. Repeated attacks of acute catarrh tend to aggravate the condition. Other things being equal, long duration and advanced age, also constitutional cachexia, render the prognosis more unfavourable.

Treatment.—The local treatment of chronic catarrh of the middle ear falls into two categories: treatment of the ear itself, and treatment of the nose and naso-pharynx.

Treatment of the Ear.—In the first place, it must be premised that treatment should commence with the permanent ventilation of the Eustachian tube. For this purpose it may be necessary to remove polypi or other growths from the nose, and even to operate on spurs or a deflected septum, which interfere with the treatment of the tube and tympanum. For diagnostic purposes, in the very rare cases in which it is impossible after the use of cocaine to pass a small Eustachian catheter through the nasal cavity, catheterisation may be performed through the opposite nasal cavity, or more effectually with my post-nasal catheter (1), which is employed with a White's self-retaining palate hook. The frequency with which inflation is repeated must be regulated by the duration of the improvement in hearing. If the air enter readily, and both ears be affected, which is usually the case, the inflation may be conducted according to Politzer's method at the patient's own home, directions being given as to how long a period it is to be used, so as to avoid over-inflation. A very useful adjunct in these cases is the employment by the patient of a chloride of ammonium inhaler, which is used twice daily for six weeks, combined with Valsalva's inflation, as described above (p. 499). After an interval of six weeks the inhaler may be used for another six weeks, and so on. By combining this with the use of the Politzer bag or catheter once or twice weekly for from four to six weeks the best results are obtained. The course of catheterisation or Politzerisation may be repeated after six months. When the Eustachian tube is impervious, inflation is assisted by the use of a fine bougie passed through the catheter, which is generally easily done. In my experience the bougie is more commonly useful as a means of ensuring the exact location of the catheter in a line with the Eustachian canal than as a means of dilating the tube itself. To-and-fro movements of the bougie when in the tube exert a beneficial massaging effect on its walls. When the bougie is well in the tube its movements can be distinctly heard through the auscultation tube.

Paracentesis of the membrana tympani is rarely required in chronic cases for mucus in the drum cavity (as distinguished from serum), as thorough inflation generally leads to better results. I have seen benefit from intra-tympanic injections *per tubam* of a weak solution of bicarbonate of sodium, where there is inspissated mucus in the tympanum, but these cases are difficult to diagnose. These injections may be tried after failure of thorough inflation in recent cases, in which there is a clear history of catarrh, while the nerve is intact and the membrane presents an opaque dull appearance. In cases of chronic middle-ear catarrh, combined with retraction of the membrane which does not yield readily on inflation, electro-massage may also be employed when the tinnitus is obstinate.

Treatment of the Nose.—Nasal and naso-pharyngeal catarrh must be treated by sniffing or syringing, with a Lynch's glass nasal syringe or a Higginson's syringe with a suitable nozzle, with a normal solution of common salt or other saline solution, and a spray of rectified spirit diluted with one to three parts of water applied to the nasal cavities. Any local cause keeping up the catarrh must be removed. Adenoid vegetations, even when small and insufficient to produce nasal obstruction, should be removed, as they help to perpetuate the catarrh. Where there is much tendency to erection of the anterior part of the inferior turbinated bodies, linear cauterisation with the galvanic cautery should be practised, as it seems to prevent the recurrence of attacks of acute catarrh. Enlargements of the posterior ends of the inferior turbinated bodies must be snared off, and other growths may require treatment. It is important in these cases to remove any nasal obstruction to free nasal respiration, but we must bear in mind that provided this can take place symmetry of the interior of the nose is of no consequence. It is in these moist catarrhal forms of otitis media that the good results on the ears of intranasal treatment are most manifest.

General Treatment.—The general treatment will be discussed under the general treatment of chronic adhesive catarrh (see below, p. 510).

Otitis Media Serosa.—Under this heading a special form of secretory catarrh of the middle ear is described, which is characterised by the exudation into the tympanic cavity of a more or less clear, limpid, yellowish fluid. If the membrana tympani be sufficiently thin this is recognised by a yellowish or reddish-yellow colour of the membrane, and when the secretion does not fill the cavity its upper edge can sometimes be seen as a more or less curved line traversing the membrane, the lower part of which is yellow and the upper part grey in colour. By placing the patient's head in the horizontal position the upper line of the liquid is still seen to retain its horizontal position, and on inflation the even surface of the fluid is replaced by bubbles, shewing through the membrane. Changes in the hearing power, according to the position of the head, also occur, but I have observed this symptom in cases in which there was no evidence of liquid in the drum. Liquid mucus in the tympanic cavity produces similar symptoms without the yellow colour, These appearances are usually seen in acute or subacute cases of middle-ear catarrh, but, as they occasionally occur in the chronic form, it is necessary to mention them here.

The prognosis in otitis media serosa is usually favourable.

The treatment of otitis media serosa in its more chronic form is very similar to that of chronic catarrh. If the ordinary remedies, especially thorough inflation with the head bent over to one side, so that the Eustachian tube of the affected ear points directly downwards, fail to relieve, paracentesis of the membrane must be resorted to. This is performed in the following manner: the auricle and surrounding parts are thoroughly cleansed, and the meatus is sterilised with a 1 in 20 solution of carbolic acid half an hour or more before the operation. Gas may be administered, or, failing this, a few drops of Gray's solution (*vide* p. 383) may be instilled a quarter of an hour beforehand with a view of anaesthetising the membrane. Then under good illumination, and using as large a speculum as possible, a vertical incision is made from above downwards into the posterior inferior quadrant of the membrane, with a small knife having a lancet- or bistoury-shaped point. If, however, the membrane bulge at any particular spot, the incision may be made at the most prominent part. An incision several millimetres long should be made and not a simple puncture, contact of the knife with the inner tympanic wall being if possible avoided. The pain of the incision is less if the point of the knife be very sharp; it is therefore advisable to examine the point with a lens before operating. The liquid is removed as far as possible by inflation or by suction with a Siegle's speculum. The ear should not be syringed. The opening generally has a tendency to close quickly, and when it does so the serous liquid may reaccumulate; on this account inflation should be persevered with for some time after the puncture, and puncture itself should only be resorted to after inflation has been given a thorough trial. Haemorrhage from injury to the bulbus venae jugularis, owing to a dehiscence of the floor of the tympanum, has been described. It should be treated by plugging the ear. When this condition can be diagnosed beforehand, Gomperz recommends a horizontal incision. Counter-irritants should also be applied over the mastoid process.

Before resorting to incision Dr. M'Bride (6) always employs massage with lanolin from above downwards behind the ear (as suggested by Ludewig), and not uncommonly has seen exudation disappear under its influence.

2. **The Adhesive Form of Middle-Ear Catarrh.**—(*Otitis media catarrhalis sicca* (Politzer); *sclerosis* (von Tröltsch); *otitis media hypertrophica* (Gruber); *proliferous catarrh* (Roosa).)

The adhesive form of middle-ear catarrh, that is, middle-ear catarrh going on to proliferation of the tympanic mucous membrane with the formation of adhesions, usually begins in the same manner as otitis media catarrhalis chronica, but its characteristic feature is that after a longer or shorter time, changes take place in the tympanic cavity which produce serious impairment of hearing, and are scarcely, if at all, favourably influenced by inflation.

Etiology.—The causes of adhesive catarrh are frequent attacks of exudative tympanic and nasal and naso-pharyngeal catarrh, paresis or paralysis of the muscles of the palate and Eustachian tube, and general diseases, such as tuberculosis, syphilis, Bright's disease, rheumatism, and anaemia. Hereditary disposition, pregnancy, and ozaena are also regarded as causes.

Pathology.—According to Politzer, in whose work further particulars may be found (12), the changes in otitis media adhaesiva may extend over the whole mucous membrane of the middle ear, or be limited to circumscribed areas. They consist of an increase in substance caused by round-celled infiltration, and in partial or total transformation of the newly-formed round cells into fibrous tissue. When new formation of connective tissue and exudation coexist, the hyperaemic, tumefied mucous membrane appears yellowish or bluish-red, infiltrated with serum, gelatinous, spongy, and evenly movable. The recesses, especially those of the fenestrae ovalis and rotunda and the attic, are filled with succulent connective tissue, which covers the stapes and the malleo-incudal body, and at times fills the mastoid antrum and cells. When the secretion has totally ceased, and complete transformation of the new-formed tissue into fibrous tissue has taken place, the mucous membrane is generally smooth, several times thicker than usual, pale, of a dull tendon-grey colour, rigid, firmly united with the underlying structures, and but slightly movable. The thickening and rigidity affect not only the lining membrane of the tympanic cavity, but also the folds of mucous membrane and ligaments which extend to the ossicles, as well as the coverings of the capsular ligaments. In yet another series of cases the tympanic cavity is traversed by numerous membranous striae and bands, which often cross each other, and by which the membrana tympani, the ossicles, and the tensor tendon are abnormally united with each other and with the tympanic walls. The bands must not always be looked upon as primary pathological formations, but as the thickened bands and folds which are so often met with in the normal ear, and which are the residue of the fetal mucous membrane cushion. The striae are sometimes transformed into osseous trabeculae by the deposit of lime salts. Localised or extensive adhesions between the membrana tympani and the inner tympanic wall, or a partial or complete obliteration of the attic or of the whole tympanic cavity, are sometimes found.

The greatest obstacle to the conduction of sound arises from the intimate attachment of the ossicles to the walls of the tympanic cavity, and from ankylosis of the ossicular joints. The adhesions are due to the formation of either a tense fibrous or osseous tissue. Ankylosis of the crura of the stapes is the most unfavourable termination of chronic inflammation of the mucous membrane of the middle ear. The ankylosis affects either the stapes alone, or the malleus and incus, with adhesions between the membrana tympani, ossicles, and inner wall of the tympanic cavity, with the formation of striae and bridges in that cavity, and with thickening and calcification of the membrane of the fenestra rotunda. Ankylosis of the stapes is favoured by congenital narrowing of the recess of the fenestra ovalis, and by the presence of those thread- and band-like filaments which are not infrequently found in great numbers in the tympanic cavity and in the above-mentioned recess. The nearer the crura of the stapes lie to the walls of the recess in the normal state, the more easily will inflammation produce ankylosis of these parts. As regards the fenestra rotunda, in normal cases threads or a fibrous network are found stretched across the recess leading to it. They are generally connected with the membrane of the round window. The fenestra rotunda is frequently found filled with a mass of connective tissue, the membrane itself thickened and covered with a villous growth and the recess greatly narrowed, or completely closed. Changes in the other articulations of the ossicles are also occasionally found.

Catarrh of the middle ear, especially if combined with chronic naso-pharyngeal affections, leads, through hypertrophy of the tubal mucous membrane and shrinking of the submucous connective tissue, to slight or marked strictures of the tube. Various other changes have been found, such as diverticula on the floor of the tube (Kirchner), infiltration of the epithelium with fat globules, hypertrophy or atrophy of the glandular layer, and infiltration of the cartilage with fat. Changes in the muscular apparatus of the tube occur, such as paralysis and fatty degeneration of the muscles in old persons. Pathological changes in the intra-tympanic muscles consist of fatty degeneration, atrophy, and cicatricial or colloid degeneration (Moos).

Examination of the Ear in these cases shews marked changes. Often there is a dry glazed condition of the meatus, with absence of cerumen. The membrana tympani is white, and thickened in patches or over its whole surface. It is often retracted as a whole, and the folds are prominent. In other cases local depressions indicative of adhesions are observed. Thinned spots (shewn by their darkness) and calcareous deposits occur, and it is often difficult, except by the history, to say whether these are due to chronic catarrh or are the result of previous suppuration in the tympanic cavity. The handle of the malleus may be apparently widened, the umbonic opacity enlarged, the light reflex either normal or irregularly ill-defined, in retraction markedly narrowed, seldom shortened or entirely lacking (13). On inflation the membrane either does not yield normally to the air-stream, or bulges into local protuberances. In many cases in my experience there is an *absence of congestion of the membrane, and especially of the manubrial plexus on inflation*, such as occurs in a normal ear. This shews that the tympanic circulation is interfered with in these cases. By the use of Siegle's speculum a local adhesion can often be made out, or the membrane as a whole may be immovable, but in these cases one must beware of a small hidden perforation, which would produce the same want of movement on applying suction.

Symptoms.—Tinnitus is very common, and may be intermittent or constant. When due to intra-labyrinthine pressure a marked diminution in the noises follows inflation or the application of suction to the meatus. Neuralgic pain in the ear, a feeling of fulness or sensation of weight in the head, dizziness and Menière's symptoms are not uncommon. As regards hearing, marked variations in the amount of deafness are not so frequent as in the catarrhal form. Paracusis Willisii, or hearing better in a noise, is common, and hyperaesthesia acoustica, or painful impressions produced by sounds, may also be present. The tuning-fork tests generally give the same results as in middle-ear catarrh.

Examination of the Nose and Throat.—In some of these cases there is evidence of old nasal and naso-pharyngeal catarrh, sometimes of a hypertrophic or of an atrophic character. In other cases the nose shews no signs of catarrh, but exhibits symptoms of a vasomotor character, with increased sensitiveness of the mucous membrane.

The prognosis is unfavourable, and the general tendency of the disease is to get worse; the special causes likely to lead to this are acute catarrh, exposure to loud noises, excessive fatigue, excesses, and old age. Serous or mucous accumulation in the middle ear may occur in the course of a chronic adhesive process.

Diagnosis.—Otitis media adhaesiva must be diagnosed from: (1) chronic moist catarrh of the middle ear; (2) otosclerosis; (3) labyrinthine affection; (4) the results of suppurative disease.

(1) From chronic moist catarrh of the middle ear it is distinguished by the absence of signs of fluid in the middle ear, and of any immediate marked improvement in hearing produced by the air douche. It seems hardly necessary to mention that in no case that is at all doubtful can the diagnosis be considered to be conclusively established unless the Eustachian catheter is passed. This can be easily done in almost all cases in adults by using a No. 1 vulcanite instrument.

(2) The diagnosis from otosclerosis (primary disease of the labyrinthine capsule) is not always easy, partly because the two diseases may coexist, but chiefly because our knowledge of the subject is still very imperfect. The diagnosis of otosclerosis rests mainly on the evidence of a normal condition of the Eustachian tube and membrane, through

which sometimes a red blush on the inner tympanic wall is seen. It must be borne in mind, however, that an apparently thickened (cloudy) membrane is not incompatible with otosclerosis, and may even occur with normal hearing. A permanently retracted membrane or a narrow Eustachian tube points more towards adhesive otitis. The tuning-fork reactions are the same in the two diseases, and are those which are usually ascribed to middle-ear disease, namely, negative Rinne, want of perception of low tones, prolonged bone conduction, normal perception of high tones, and in unilateral deafness the fork by Weber's test referred to the affected ear. Möller, however, states that he finds generally in otosclerosis diminished bone conduction and the lower limit of hearing comparatively slightly raised, much less than in chronic catarrh, whilst the upper limit is in almost all cases markedly lowered. In the latter statement he is supported by Blegvad and by Burger and Zwaardemaker, who have also noticed repeatedly in otosclerosis that the handle of the malleus is unusually white and angular, and has on it little punctiform exostoses. In both these diseases the deafness probably has a tendency to gradual increase, but a rapid onset of the deafness would favour otosclerosis, likewise early implication of the labyrinth. Any history of previous attacks of acute otitis, and of course of previous catarrhs, would be in favour of the case being one of adhesive catarrh rather than primary disease of the labyrinthine capsule. Dr. Thomas Barr recommends exposure of the head of the stapes by forming a window-opening through the tympanic membrane, and observing through it the condition of the crura, and determining, by pressure with the feather probe of Lucae, the mobility of the stapes, as a means of distinguishing fixation of the stapes due to binding of the crura from that due to rigidity of the annular ligament.

(3) We assume that there is a complication of labyrinthine disease if, with symptoms in the membrana tympani of chronic adhesive catarrh, there is diminished bone conduction as compared with that of a normal ear, together with marked deafness and continuous noises. Diminished hearing power for high notes, and, in unilateral deafness, the fact that the tuning-fork is heard in the good ear by Weber's test, also point to nerve deafness. In my experience, in a large proportion of the cases of chronic deafness associated with thickening of the tympanic membrane (apart from those due to old age) both air and bone conduction are diminished. The distinction from a case of pure nerve-deafness is made by the absence of tympanic and Eustachian tube lesions, and by the tuning-fork tests above mentioned. In doubtful cases, which sometimes give mixed tuning-fork reactions, Hartmann's test of the gradual diminution in the length of time the tuning-fork is heard as the scale is ascended may help to distinguish labyrinthine cases. Gellé's test may also be used to distinguish between fixation of the stapes (as occurring in advanced otitis media adhaesiva and in otosclerosis) and nerve-deafness. Opinions, however, differ as to the value of this test. Although much has been done already, the whole subject urgently needs further light thrown on

it by the pathological investigation of cases which have been carefully examined during life.

(4) The results of suppurative disease, when there is no perforation present, must be distinguished from otitis media adhaesiva. This is done in the first place by the history of suppurative otitis, and when this cannot be relied upon, by the appearance of the membrane, shewing cicatrices, adhesions, and so forth, although these are not always easy to distinguish from atrophic patches occurring in otitis media adhaesiva. The deafness is generally not progressive in the "results of suppuration," and the subjective tinnitus is also apt to be less marked.

Treatment.—Local Treatment.—In this disease treatment is directed not only to improving the hearing, but to preventing any aggravation of the deafness, while endeavouring to mitigate the tinnitus and the other symptoms mentioned above. In the first place, inflation of the ears must be practised in order to ascertain that the Eustachian tube is pervious, and if possible to stretch or break down any intra-tympanic adhesions. A narrowed or impervious tube must be treated by catheterisation, if necessary with the use of a bougie. Inflation, whether with the catheter or the Politzer bag, must be carefully performed, or it may aggravate the deafness. A distinct lasting improvement in hearing is a favourable prognostic sign, but in many of these cases not only does inflation produce no improvement, but even causes temporary aggravation of the deafness, to be followed later on, however, in some cases by a moderate amount of relief to the symptoms. In practice, I find that three or four successful inflations with the catheter at intervals of a week or a fortnight are sufficient to make a decided prognosis, and to settle, from the presence or absence of improvement, whether it is advisable to continue them. If producing distinct improvement, they may be used once or twice a week, but not for more than four to six weeks. In any case, the effect of the inflations should be carefully watched, lest they aggravate the noises or deafness. When catheterising in this disease, as well as in chronic middle-ear catarrh, I am in the habit of filling the bag with vapour drawn from a bottle half-filled with a mixture of one part chloroform and two parts sulphuric ether.

Many aurists combine these inflations with injections *per tubam* into the middle ear. The solutions most commonly used are bicarbonate of sodium, hydrochloride of pilocarpine, chloride of ammonium, and iodide of potassium (about 1 per cent solution). Of any of these 5 to 6 drops (sterilised) are injected lukewarm into the catheter after it is in position, and blown gently up the tube with the bag. They may also be injected through a fine tube passed through the catheter and up the Eustachian tube. Intra-tympanic injections of sterilised liquid vaseline (10 to 15 drops warmed) were strongly recommended by Charles Delstanche. After due trial I have renounced these injections as having no special value, the only exception being, as mentioned on p. 502, injections of bicarbonate of sodium, which seem useful in cases in which there is reason to suppose that there is inspissated secretion in the tympanum.

In addition to inflation the effects of massage of the membrana tympani should always be tried. This is best applied with an electric massage apparatus. The strokes of the piston should be very short at first, about a third of an inch in length, and the application not longer than from half to one minute. Subsequently the stroke can be lengthened, and the duration of each sitting extended to three minutes. The membrane must be watched through a Siegle's speculum during the massage. The massage, in my experience, has more effect on the noises and disagreeable head symptoms than on the hearing power. The patient often expresses himself as partly or entirely relieved of the tinnitus, and says that the ear feels clearer directly after the massage. The frequency of its application must depend on the duration of this relief. Intelligent patients can be taught to apply the massage themselves, under direction as to frequency and duration. In case of relaxation or thinning of the membrane it must be used with great care. While these means are being employed the patient may use a chloride of ammonium inhaler in the manner described above (p. 499), but it is not likely to have the same beneficial effects that it has in cases of moist catarrh. The inflation of other vapours into the tympanum has been recommended by different authors, but I have not found them of any special advantage, and therefore do not use them. I have no practical experience of Lucae's pressure probe. Subcutaneous injections of fibrolysin have recently been recommended as an adjunct to the mechanical treatment of these cases.

Operative measures on the middle ear have been recommended and practised chiefly by Continental observers. The chief are: the division of the posterior fold in cases of marked retraction, division of the tensor tympani (Weber-Liel), excision of the membrane with the malleus and incus, excision and mobilisation of the stapes, and the severance of adhesions. The formation of an artificial perforation in the membrane in these cases may considerably improve the hearing, but the difficulty has always been to maintain the opening, which is very prone to close. Even when the malleus is excised at the same time the gap is generally soon closed with a cicatrix, rendering the effect of the operation nugatory. The indications for these operations are so indefinite, and the published results are so unsatisfactory, that it may be said, with the possible exception of the division of the posterior fold, that none of them have met with any general acceptance.

Treatment of the Nose.—Much must not be expected from the treatment of the nose and throat in these cases. Catarrh, if present, must be combated by the ordinary means, but in cases of nasal obstruction no directly beneficial effect on the ears from operative measures must be expected unless the hearing improves markedly on inflation. In all other cases the nasal obstruction should only be operated on if it is the cause of naso-pharyngeal or nasal catarrh, or produces other than ear symptoms which require it. The moot question of operating on nasal obstruction with a view *to preventing the deafness in otitis media chronica adhaesiva from* *increasing* is one on which it is better to err by doing too little than too much, as it is not likely that in a confirmed case of this character the nasal obstruction makes the hearing worse, except indirectly by maintaining nasal catarrh.

The general treatment of these cases, as well as of the secretory form of catarrh, consists in doing everything possible to avoid a fresh attack of acute catarrh. The patient's residence should if possible be in a dry elevated position, and a visit to Switzerland sometimes has a beneficial effect, though it cannot be said to be of permanent benefit in adhesive catarrh. The ordinary rules for prevention of catarrh, such as exercise, fresh air, avoidance of cold or damp feet, warm baths, should be recommended, but cold baths appear to have a bad effect in cases of adhesive catarrh (14). Over-fatigue is deleterious; sitting up late at night therefore should be eschewed, and especially night-nursing. It must be remembered that the strain of listening to ordinary conversation is often very great in these cases. When anaemia is present iron should be administered, and in most cases of adhesive catarrh strychnine is indicated. If there is any suspicion of syphilis mercury and iodide of potassium should be carefully administered in addition to the local treatment. Excessive use of tobacco should be avoided, likewise exposure to loud sounds. Quinine, if taken at all, should be in small doses only.

In cases, apparently unconnected with syphilis, in which the nasal cavities are unusually dry small doses (gr. iii. to gr. v.) of iodide of potassium, with spiritus ammoniae aromaticus, ♏ x., aq. ad ℥i., to be taken hot after meals twice or three times a day, sometimes have a beneficial effect on the hearing. The mixture causes a flow of mucus from the nose, which the patient should be prepared for. If it produce no benefit in four weeks it should be discontinued. When tinnitus is troublesome bromide of potassium or ammonium may be added to the mixture with advantage. It is probably in gouty cases that this mixture answers best.

In pulsating tinnitus, which may occur in either of these classes of cases, dilute hydrobromic acid in ʒss. to ʒi. doses, combined with strychnine, sometimes has a markedly beneficial effect, at other times it fails entirely to give relief. Stimulating drops, such as ether sulph. ʒss., tinct. valer. ʒi., glyc. ʒvi., ft. guttae pro auribus, often have a beneficial effect on the tinnitus. Four drops are either painted into the cartilaginous membrane with a small brush, or inserted on cotton-wool and kept in for ten minutes twice daily.

In advanced cases, in which intercourse with the patient is very difficult, the patient may be tested with different forms of ear-trumpets and tubes (*vide* p. 552). Several forms of electrical apparatus are on their trial, but unfortunately up to the present we have no small invisible tube, such as patients are always asking for, which is of any real benefit.

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E. C. B.

OTOSCLEROSIS

By ALBERT A. GRAY, M.D.

THERE is no doubt that the "progressive deafness" of the older writers indicated roughly cases now known as examples of otosclerosis. As knowledge increased a distinction was made between deafness due to changes in the middle ear and that due to changes in the labyrinth. At this period the disease previously called "progressive deafness" became known as "dry catarrh of the middle ear," owing to the mistaken assumption that the middle ear was the seat of the morbid process. The name was most unfortunate, since the affection is not particularly dry, is not a catarrh, and it is not of the middle ear. The mistake was rectified by careful pathological investigation, which shewed that the origin of the symptoms was in the bony capsule of the labyrinth. The disease was then spoken of as otosclerosis, osteoporosis of the labyrinthine capsule, or rarefaction of the labyrinthine capsule.

Otosclerosis may at present be defined as an affection characterised by progressive deafness and other symptoms, without the presence of serous or purulent discharge; and, pathologically, by the presence of changes in the bony capsule of the labyrinth, causing in all or most cases ankylosis of the stapedio-vestibular synostosis.

History.—Valsalva first shewed that deafness was sometimes due to fixation of the stapes in the oval window. Morgagni and Meckel also observed the condition. These observations, however, were made in isolated cases, and for many decades no further advance of any importance is to be recorded. Toynbee at length, as the result of the examination of numerous cases, proved that the condition, far from being rare, was extremely common, and, had the methods of clinical and pathological research then available been more accurate, it is probable that an investigator possessed of such patience as Toynbee displayed would have solved the problem, at least to a great extent. The time, however, was not ripe, and even when pathological and clinical methods became more perfect there was no investigator of sufficient originality and independence of spirit to elucidate the matter. It was assumed, and quite unjustifiably, that fixation of the stapes was only one of the results of inflammatory action in the middle ear. The first suspicion that this assumption was incorrect arose in 1861 when Moos published the results of a careful examination of the temporal bone in which fixation of the stapes was found without any pathological changes in the middle ear. In 1885 Bezold was enabled to associate the clinical features of the disease with the pathological changes, and further examinations by other observers revealed similar conditions. By means of tuning-fork and other tests the difficulties of diagnosis have, to a large extent, disappeared, but unfortunately the advance in therapeutics has been very small.

Etiology.—*Age.*—It is commonly stated that otosclerosis begins most frequently in middle life. It rarely comes on before twenty or after fifty-five. According to Panse it most commonly begins during the fourth decade, but it must be remembered that with a disease of such insidious onset there is probably a slight diminution in the hearing power for a long time before the patient is aware of it. This is particularly the case when, as frequently happens, one ear is affected first. We may therefore conclude that the disease most commonly affects those between the twentieth and fortieth years.

Sex.—The affection is more common in women than in men. Bezold found that about 60 per cent and Denker that 58 per cent of the cases are in women.

Heredity plays an important part in the incidence of the disease. Körner relates an example in which two victims of otosclerosis married and had twelve children, and all of the twelve were deaf. Denker gives 40·5 per cent, Bezold 52·0 per cent, and Siebenmann 35·0 per cent as the proportion of inherited cases. My experience agrees more closely with that of Siebenmann, and I would put the percentage even lower. It must be remembered that it is difficult to prove the influence of heredity in many cases. The statement of a patient that his parents, grandparents, or other relatives were deaf is of little value, since it is frequently found on investigation that the deafness was due to previous or existing suppuration which could have nothing to do with the inheritance of otosclerosis. Still it must be admitted that after eliminating all doubtful cases, inheritance plays an important part in the occurrence of the disease. According to Körner women transmit the tendency more frequently than men, besides being themselves more liable to suffer from it.

I do not know of any investigations as to the influence of race upon the occurrence of otosclerosis, but if inheritance plays such an important part as some observers affirm it would naturally be expected that race would also have some effect upon the incidence of the affection.

Morbid Anatomy.—In considering the pathological aspect of the disease a careful distinction must be drawn between the morbid anatomy and the pathogenesis. The first deals with matters of observation, whilst the latter is for the most part speculative. The great recent advance of knowledge regarding the anatomical changes is due to Katz, Politzer, Siebenmann, Bezold, Habermann, Panse, and others.

The disease begins by the absorption of bone in some part of the capsule of the labyrinth. The region which is supposed to be most commonly affected is that immediately above and in front of the oval window. Pathologists may perhaps have been too ready to assume that this is the part most commonly attacked. In the cases that have been examined no doubt it is so, but this may merely be because when the anatomical change takes place at this part it causes deafness, and because it is only in cases presenting this symptom that an examination is made. We cannot definitely say, therefore, whether the disease in the bone invariably causes symptoms of which the patient is cognisant, and it is quite possible that there may be cases in which the bony change is limited to portions of the labyrinthine capsule in such a way that no physiological function is sufficiently disturbed to affect the patient consciously. Any region of the capsule may be affected as, for example, the porus acousticus, the modiolus and spiral lamina, the edges of the round window, or the walls of the semicircular canals. It is not, therefore, difficult to understand that the symptoms of the disease may vary considerably.

The pathological process consists essentially in absorption of the old bone followed by the deposition of new-formed spongy bone in its place. The diseased focus is at first sharply differentiated from the surrounding healthy bone by a line of demarcation. The Haversian canals within this area become enlarged, their walls being absorbed by osteoclasts. After this has occurred new blood-vessels are formed, osteoblasts make their appearance, and new bone is laid down which has a great affinity for staining reagents, being in this respect markedly different from the original bone. The new-formed bone gradually becomes more and more dense, the medullary spaces diminish in number and size, and the new-formed blood-vessels become smaller in diameter until the bone is again compact. It gradually loses its affinity for staining reagents and, indeed, can no longer be distinguished from the old bone surrounding it. In the process of absorption the cartilage lining the oval window and the footplate of the stapes, along with the adjacent portions of the annular ligament, disappear and are replaced by bone. Thus it is that the stapes becomes fixed. There are great variations in the extent to which the stapedio-vestibular synostosis is affected by the process. Sometimes there is merely a thin lamella of bone joining the stapes to the margin of the oval window, whilst in other cases the walls of the oval window, the annular ligament, and the stapes are united into one bony mass. Small exostoses are occasionally found in the margin of the round window, and the oval window is sometimes affected in the same way. According to Katz these changes are not confined to the capsule of the labyrinth, but may be found in the incus and malleus.

In this respect I agree with Katz, and in the case which I recorded not only were the malleus and incus affected, but the bones of the skull in general were remarkably soft and may also have been implicated in the process, but no microscopic examination of them was made.

It is important to observe that during the whole process there is no sign of inflammatory action.

Pathogenesis.—While the anatomical changes just described are definitely ascertained, the physiological disturbance by which these changes are brought about is not known. Certain speculations on this subject have been made, but space does not permit of their full consideration. The various hypotheses will, therefore, be described as shortly as possible, without much comment as to their relative merits.

Habermann and Katz believe that the disease is due to infection from the middle ear. There is but little evidence to support this, and in the majority of cases the middle ear is normal. Siebenmann is of opinion that otosclerosis is hardly a disease at all, but is really a process of ossification occurring in the layer of cartilaginous cells immediately outside the labyrinth. This layer does not undergo ossification as in other bones, but remains as cartilage throughout life. If, however, for any reason the layer does undergo ossification, then otosclerosis is the result. This hypothesis is satisfactory in that it accounts for the absence of any sign of inflammatory action during the course of the disease, but on the other hand it does not explain the occurrence of pathological changes in such places as in the malleus and incus. Malherbe, relying on the investigations of Poncet, made the interesting suggestion that otosclerosis is, in many cases at least, a manifestation of tuberculosis. In support of this he refers to cases in which evidence of previous tuberculous disease was present, either in the patient himself or in near relatives. My own suggestion is that the foci of disease in the temporal bone represent a process in which minute portions of bone have died from insufficient nourishment, and are replaced by new bone. In this process the cartilage lining the oval window also dies, because in this, as in other regions, articular cartilage is nourished from the bone beneath. Inflammatory action is absent, because infection does not occur, the whole process being comparable to that which occurs in the absorption of an aseptic infarct. In support of this opinion is the existence of a sharp line of demarcation round the affected part and the absence of inflammatory action. For more detailed consideration of this interesting subject the reader may be referred to the papers by Körner, Malherbe, Siebenmann, and myself, and to the monographs of Panse, Denker, and Bezold.

Before leaving this part of the subject it is necessary to refer to the constitutional conditions which have been supposed, rightly or wrongly, to be associated with otosclerosis. No really satisfactory evidence has been put forward to prove any relationship between otosclerosis and tuberculosis or syphilis, and the same may be said of that heterogeneous collections of symptoms vaguely termed rheumatism. Pregnancy and the puerperium undoubtedly make the symptoms worse if these have already made their appearance, but they cannot be admitted as direct causes in the occurrence of otosclerosis. Anaemia has more claim to consideration as a factor in causation. Dr. Dundas Grant and I have both observed that otosclerosis frequently begins in young women when suffering from chlorosis. This would, moreover, explain why the disease is more common in women than in men. It must be admitted that no known constitutional condition, either inherited or acquired, is always present at the onset of otosclerosis; and we are almost driven to explain its occurrence by supposing that any factor which lowers the general tone of the system may be the exciting cause of otosclerosis in those who are disposed to the disease, either by inheritance as suggested by Körner, or in the way I have suggested. Beyond this it is hardly possible to go until more data have been ascertained upon which a broader basis for speculation may be formed.

Symptoms.—*Dulness of hearing* is the most important symptom of otosclerosis, though it is not always the most distressing. So gradual is the onset of the deafness that the sufferer is usually very indefinite in his statements as to the time at which it first became noticeable. Deafness is also, in the great majority of cases, the first symptom, but occasionally tinnitus precedes it. The increase in the dulness of hearing is very slow, and it may be ten or twenty years before the loss becomes stationary. The ultimate degree of the deafness may be either comparatively slight or very great, but only in rare cases does it become so marked that the patient cannot understand a loud conversational voice in close proximity to the ear. In such cases it may be assumed, with a fair degree of certainty, that the auditory nerve or the structures within the labyrinth have been affected. In some cases a sense of fulness in the ear is complained of, but pain is conspicuous by its absence, and even when present has probably no relation to the disease.

Second only in importance to the deafness are the subjective sensations of sound by which the sufferers from otosclerosis are so frequently affected. In a few cases the *tinnitus* is more distressing than the deafness. The symptom is present in two-thirds of the cases, but fortunately in only a small proportion of these is it so severe as to affect the patient seriously. Attempts have been made to classify the various kinds of subjective noises with a view to bringing about a more accurate diagnosis, but hitherto without success. In some cases the tinnitus is intermittent, in others it is continuous, and in all the intensity varies in degree. Bodily or mental exhaustion are among the most potent factors which bring it into prominence. Want of mental occupation also tends to make it more noticeable, and if the attention be concentrated on any extraneous subject it becomes less distressing. It is probably for this reason, as well as owing to the surrounding silence, that many complain of tinnitus only after going to bed at night. Sudden changes in the calibre of the blood-vessels affect the patients unfavourably; even movements from the recumbent to the erect posture frequently accentuate the trouble temporarily, owing to changes in the flow of blood through the labyrinth. Alcohol and tobacco, even in small amounts, increase the intensity of the noises, and the patient is wise to renounce them altogether. Fortunately, most patients become accustomed to the noises, and the symptom loses its worst feature, that of seriously irritating the sufferer. There are some cases, however, in which the tinnitus seems to increase, or at least to bulk more and more largely on the patient's mental horizon. To such an extent may this occur that the lives of these individuals are a constant burden to them, and they may be quite unable to concentrate their attention upon their work. Suicide has been resorted to as an escape from the intolerable condition of life, and I know of at least one case in which it brought an apparently sane individual to the asylum. Temperament undoubtedly plays a part in the story of the troubles which the patient unfolds to the physician. Those of an optimistic and objective turn of mind complain but little, whilst the atrabilious and introspective may be affected more by this symptom than by the deafness. In this connexion it may be observed that children seldom or never complain of tinnitus, but it must be remembered that otosclerosis is almost unknown in childhood ; and it is precisely in this disease, above all others, in which tinnitus is apt to occur in its most distressing form. It is said that, with the onset of complete deafness, the symptom disappears, but such a conclusion is rare in otosclerosis, and in a case in which the deafness reached a higher degree than in any other I have ever seen, the tinnitus was extremely distressing.

The direct cause of tinnitus is obscure. It has been suggested that it indicates disease of the labyrinth or auditory nerve, but this is almost certainly incorrect. Moos thought that it may sometimes be due to calcareous deposits in the auditory nerve, and I have suggested that it may occasionally be called into existence by the deposit of these salts in the basilar membrane. That such deposits do occur is undoubted, for I have been able to demonstrate this in a case of otosclerosis in which tinnitus had been complained of during life (4). But these are isolated examples, and the explanations given probably do not apply to the majority of the cases, since the tinnitus frequently occurs early in the disease before any change in the labyrinth or nerve is likely to have begun. Siebenmann accounts for some of the sudden noises, which are heard occasionally by the sufferers from otosclerosis, by supposing that a rupture takes place in the thin membranes which separate the intra-labyrinthine fluids from the lymph-spaces of the diseased bony foci, but obviously this explanation applies to a very small proportion only of the cases. Habermann explains the occurrence of the noises by assuming that the nerve-trunks are implicated in the bony changes. The frequency of the symptom, however, demands a more constantly present factor or set of factors than those given above, and it appears to me that the symptom probably arises from the change in the physical conditions to which the intra-labyrinthine fluids are subjected. Fixation of the stapes must, to a certain extent, interfere with the compensation required to allow for the changes of tension to which these fluids are subjected during the cardiac systole and diastole. Hence the changes of pressure during the phases of cardiac activity will be brought to bear upon the organ of Corti more than in normal circumstances, and the consequent stimulation of that structure will produce the subjective sensation of sound. These various explanations, it must be remembered, are only speculative, and it will be a long time before definite proofs can be given in favour of the correctness of any of them. It is, however, important to confute the common error to the effect that tinnitus has its origin in the central nervous system. There is no evidence in favour of such a view that will bear investigation. It will, of course, be understood that the hearing of voices and musical melodies are not referred to in this connexion, as these symptoms are only met with in the insane (*vide* also p. 370).

Vertigo is rare in otosclerosis, occurring, according to Panse, in about 9·5 per cent only of the cases. It is doubtless due to implication of the cristae acousticae of the vestibule and canals or of the nerves supplying these structures, or in some cases to calcareous masses in these structures. In a case recorded by me, in which giddiness had been complained of during life, a large mass of this nature was found in the vestibule. It is interesting to note that the implication of portions of the canals other than the ampullae does not seem to cause vertigo, for in another case which I examined the posterior canal was almost obliterated at two places, although there had been no complaint of giddiness during life.

In cases of fixation of the stapes the curious symptom known as *paracusis Willisii* is frequently present. It is impossible to discuss the cause of the symptom here, but it should be noted that it only occurs when the deafness is bilateral and considerable in degree. It is, moreover, an unfavourable symptom (*vide* p. 370).

In the vast majority of cases of otosclerosis both ears are affected, though the symptoms may make their appearance in one organ before the other is at all defective. It is usually stated that about 80 per cent of the cases are bilateral; but in all probability the percentage is even higher than this, because in any list of statistics some of the 20 per cent of the cases reported as unilateral would ultimately become bilateral.

In many individuals who have been deaf for a long time a change in the physiognomy is frequently to be noted. For several years after the onset of the deafness a strained look is usually, though not always, present, and this is apt to be accentuated if tinnitus is a marked feature. As time passes, and particularly if the deafness increase and the tinnitus become less irritating, the strained look gradually passes into one of calm serenity. When the constant effort to listen has been given up, and the emotions are no longer constantly stimulated through the sense of hearing, the expression gradually becomes calm and placid.

Signs.—In uncomplicated otosclerosis the mucous membrane of the tympanum is usually unaffected, or at the most there is a slight degree of hyperaemia over the promontory. Consequently the drumhead either presents a normal appearance or shews a slight rosy tint in its lower portions. The various changes in the membrane which were supposed to be characteristic of dry catarrh of the middle ear, such as thickening, opacity, indrawing and so forth, were errors, due in part to ignorance of the great variations in appearance which are quite consonant with a healthy membrane and in part to subserviency to authority. Even the deposit of calcareous salts in the membrane, although undoubtedly pathological, is probably only a coincidence in otosclerosis. It can at least be said that these deposits are not at all uncommon in subjects with perfectly normal hearing. The only sign, on inspection of the membrane, that is of any value in the diagnosis of otosclerosis is the rosy tint referred to above, and in regard to this it may be said that the condition is by no means so common as might be inferred from the statements made in text-books. The membrane in otosclerosis is usually normal, and this point is of help in the diagnosis.

Diagnosis.—The diagnosis of otosclerosis is made by a consideration of the signs and symptoms described above, aided by certain tests for hearing and by the results obtained from inflation of the middle ear by means of the catheter.

On inflation of the middle ear the air is heard through the auscultation tube to rush up with a normal sound, provided there be no coincident catarrh of the Eustachian tube or of the middle ear. After inflation in cases of fixation of the stapes there is either no improvement in the hearing, or at most an insignificant amount.

The tests for hearing are carried out by means of tuning-forks, Galton's whistle, the watch, and the voice, both conversational and whispered. Testing with the voice is simply done for the purpose of ascertaining the distance at which it may be heard. By constant practice the aurist learns to pitch his voice, whether whispered or conversational, at approximately constant degrees of intensity. Testing with the watch is done by finding the distance at which the patient hears the tick, and comparing it with that at which it is heard by a normal ear. Attempts have been made to replace the watch by instruments known as acoumeters, but the use of these has never been very much encouraged, at least in this country, and they may be dispensed with.

The tests just described merely give information as to the hearing power of the patient, without regard to the cause of the deafness. In order to discover the latter, resort must be had to a special form of tests known as the tuning-fork tests.

Rinne's Test (*vide* p. 356).—In fixation of the stapes we find usually, but not always, that the test gives either a negative Rinne or a diminished positive Rinne. In serious affections of the labyrinth there may be an increased positive Rinne, and the result, so far as diagnosis of fixation of the stapes is concerned, is valueless.

Gellé's Test (*vide* p. 357).—According to Gellé, if the stapes be fixed there will be no difference in the intensity of the sound during compression and relaxation, whereas if the stapes be free the sound will be diminished during compression, as in a normal ear.

In Schwabach's test the duration of bone conduction in the patient is compared with that in a person with normal hearing. If the bone conduction is longer in the patient, then he is presumed to be suffering from middle-ear disease or fixation of the stapes.

In all these tests the underlying principle is the same, namely, that of the relative duration of sound by bone conduction. It is important that the aurist should bear this in mind, and not attribute to each individually the same value which is to be attached to two or more tests, each depending upon a different principle. Thus, it is but little confirmation in diagnosis to find that Rinne's, Gellé's, and Schwabach's tests agree in pointing to middle-ear disease or fixation of the stapes. It would rather be remarkable if they did not so agree. On the other hand, great importance would be attached to the observation that Rinne's test agreed with Bezold's in pointing to middle-ear disease or fixation of the stapes, for Bezold's test in no way depends upon the conduction of sound by the bone.

In carrying out the tests described above it is desirable that the tuning-fork employed be of comparatively low pitch, otherwise the results may be ambiguous.

Testing with Notes of Different Pitch (see p. 351).—Although loss of hearing for the low notes is always present in cases of fixation of the stapes, it must be admitted that a small portion of the upper end of the scale is frequently inaudible to the patient. There is as yet no direct proof that this is always due to a coincident affection of the labyrinth; but if the portion inaudible at the upper end of the scale be at all extensive, it is fair presumptive evidence that that organ is disordered.

Bezold has grouped together the results of the tuning-fork tests and formulated a symptom-complex consisting of these three conditions—prolonged bone conduction, negative Rinne, and loss of hearing for the low notes. These are known as "Bezold's triad of symptoms"; and the originator maintains that, when the three are all present, fixation of the stapes may be diagnosed with certainty, provided that suppurative and catarrhal conditions in the middle ear and Eustachian tube have been excluded. There can be little doubt that Bezold is justified in his opinion, since in every case in which the diagnosis has been made during life and the ear has been examined after death, fixation of the stapes has been present. It is unfortunate, however, that in many cases of undoubted otosclerosis the triad of symptoms is incomplete. Rinne's test is not always negative, and the bone conduction is not always prolonged. This is the result of changes in the labyrinth, or in the auditory nerve in its bony channels. But even when these tests are incomplete the difficulty in making the diagnosis is not so great as might appear. The history of the case, the bilateral nature of the affection, the tinnitus, the paracusis, and so forth make up a clinical picture unfortunately too well known.

Now it must be remembered that the diagnosis arrived at by the means above described is not a diagnosis of otosclerosis, but of fixation of the stapes. All the signs and symptoms mentioned might be, and undoubtedly sometimes are, due to fixation of the stapes, the result of existing or previous suppurative conditions in the middle ear. The diagnosis of otosclerosis is made by the exclusion of such changes as shewn by inspection of the membrane. It is, of course, quite possible that otosclerosis might supervene in an ear in which suppurative changes had occurred, and in such cases it would be impossible to make a differential diagnosis with any degree of certainty.

Prognosis.—In spite of the poor results of treatment the disease tends ultimately to come to a standstill while there still remains sufficient hearing power to enable the patient to understand a loud conversational voice at a little distance from the ear. There are certainly exceptions to this rule, but they are not very common. There is, however, one point about which the patient should be warned. Senile deafness, which is due to changes in the labyrinth or auditory nerve, makes its appearance earlier in those who are already the subjects of otosclerosis. Thus, an individual in whom the deafness due to otosclerosis has not made any progress for fifteen, twenty, or even thirty years may find that at about the age of sixty or sixty-five his hearing is again getting worse. It is therefore wise to advise these patients so to order their lives that their burden may press on themselves and their friends as lightly as possible in the years "when the daughters of music shall be brought low." They must cultivate interests which are, so far as possible, unaffected by the loss of human intercourse through the medium of the voice.

The question of marriage may arise, and on this point opinions are divided. Körner is opposed to the marriage of the female members of a family in which there is a tendency to otosclerosis, the females being selected both because they are more liable to transmit the tendency, and because pregnancy and the puerperium have a deleterious effect upon the course of the disease. The responsibility of giving advice upon this subject does not, however, arise very frequently. In giving an opinion the physician must view the subject in a broad light. Distressing as otosclerosis is, the disease does not prevent the fulfilment of a happy existence, and there have been many useful lives in spite of deafness in husband or wife.

In the matter of pregnancy also the physician must be careful not to look at the matter from too exclusive a standpoint. It is certainly true that pregnancy and the puerperium may have a bad effect upon the course of the disease, though this is not always so apparent as some writers suppose. But it is open to question whether the possible diminution in the hearing power is not fully compensated for by the gratification of the maternal instinct, and he is taking a grave responsibility upon himself who advises the repression of the latter. Perhaps the wisest course to pursue in regard both to the question of marriage and child-bearing is to point out to those interested the risks undertaken, and then leave them to decide the matter for themselves. So far as the transmission of the tendency to otosclerosis in the offspring is concerned, it should be remembered that the affection does not make its appearance until adult life is reached. Thus, even if such offspring should become affected, their education, both general and technical, will not have been interrupted by deafness.

Treatment.—The treatment of otosclerosis is unsatisfactory. No drug hitherto discovered will dissolve bone in such a way as to leave other tissues unaffected, and it is therefore impossible to mobilise the stapes by any internal medication when there is true bony ankylosis. In recent times thiosinamin and fibrolysin have been employed in order to soften and cause absorption of connective-tissue bands which may interfere with the mobility of the stapes. Impartial investigators have not been impressed by the results obtained in this way. It is unnecessary to give the list of useless drugs which have been recommended for the same purpose, either locally through the catheter or generally through the circulation.

This list includes phosphorus, which has been vaunted in recent times, and the surgeon is well advised not to trouble the patient by prescribing it.

Although it is hopeless to expect any drug to produce an effect upon the rigidity of the stapes, it is of the utmost importance to try by every means to improve the general health. Above all, the serious mistake of not recognising the presence of chlorosis, even in its slightest manifestations, must be avoided. This is not only the most serious, but also the most common mistake. With the cure of the chlorosis a slight improvement in the hearing usually occurs, not, of course, on account of the effect of the drug upon the fixed ossicle, but because of the improvement of the circulation within the labyrinth. Furthermore, if the anatomical changes in the capsule are sometimes due to anaemia, the cure of the chlorosis may prevent the further advance of the disease. Strychnine occasionally acts beneficially, probably also on account of its effect upon the circulation, but the improvement only lasts with the administration of the drug.

The tinnitus is almost as difficult to treat as the deafness, and numerous drugs have been tried with more or less success. Perhaps the least useless of these are the bromides given in fairly large doses. Quinine has also been tried, but its administration must be carefully watched, since it may cause the very symptom under discussion. Potassium iodide and nitroglycerin are occasionally useful, but no rule can be laid down as to the cases in which these drugs may prove beneficial, and the surgeon must satisfy himself by trial as to which gives most help in the individual case.

Much more important than the administration of drugs is the avoidance of alcohol and tobacco. These are always deleterious in the disease, even in such moderation as would have no noticeable effect upon the hearing in healthy persons.

Exhaustion, whether mental or physical, has a very bad influence on otosclerosis, and should always be avoided.

Surgical intervention has proved equally unavailing in the treatment of otosclerosis. Inflation of the tympanum by means of the catheter, with or without the addition of chemical agents, is not only useless but occasionally harmful. There may be exceptional cases in the early stages in which inflation may loosen the ossicle, but even in such the possibility of error in diagnosis must be remembered, and it is probable that a number of cases of recorded improvement have been in reality examples of fibrous adhesions in the region of the stapes due to inflammatory changes in the middle ear. The effect of rapid vibrations upon the drumhead by means of the various pneumo-massage instruments is uncertain. There are some cases which present all the signs of otosclerosis, and yet appear to benefit, to a certain extent, by this treatment. The improvement is certainly never very great, and can hardly be due to mobilisation of the adherent stapes. Such benefit as may accrue is probably due to the beneficial effect of the massage through the round window upon the blood-vessels of the labyrinth. Be this as it may, it is wise to give the method a trial, since, if improvement does not result at once, it will not appear at all, and time is not wasted nor are false hopes entertained. Furthermore, the tinnitus is sometimes relieved by the massage even when the hearing is not improved; but unfortunately the relief from the tinnitus is not often obtained in the very cases in which it is most desired, that is, in the worst ones.

There are few conceivable operative procedures which have not been attempted for the relief of otosclerosis. None of these have been found to be successful. The stapes has been mobilised and removed, with only temporary benefit, or, more often, with none at all. The tympanum has been eviscerated, the labyrinth has been opened and even portions of it have been removed, but the deafness obstinately remains and the tinnitus is as loud as ever. Even the auditory nerve has been cut within the cranium for the purpose of relieving most distressing tinnitus, but unfortunately the symptom returned as bad as before, and the deafness was not improved. A satisfactory method of treating otosclerosis is still to seek.

In conclusion, it must be impressed upon the surgeon that before giving up hope he must be absolutely sure that the case in hand is really one of otosclerosis. Every aurist occasionally sees cases which have been given up as suffering from fixation of the stapes, but which prove, on careful examination, to be examples of Eustachian catarrh or fibrous adhesions among the ossicles.

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AFFECTIONS OF THE AUDITORY NERVE AND LABYRINTH

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ALTHOUGH much laborious work has been and is being done in connexion with the pathology of the internal ear, we cannot yet consider our knowledge sufficient to justify any attempt at a pathological as opposed to a purely clinical classification. Space does not permit of any lengthy physiological discussion as to the functions of the internal ear. For our purpose it will be sufficient to bear in mind that the auditory nerve consists of two main parts, namely, (1) that distributed to the cochlea, (2) that which supplies the semicircular canals and vestibule. It is now generally assumed that the cochlea is concerned mainly, if not altogether, with the act of hearing, whilst the semicircular canals have a very definite relation to the faculty of equilibration. It follows from this that when—whether in its trunk or peripheral distribution—the cochlear portion of the nerve of hearing is affected, interference with that sense is to be looked for, whereas when the vestibular part is implicated, we must expect disturbance of equilibration.

Let us now consider the symptoms produced respectively by the cochlea and semicircular canals a little more in detail. Although not absolutely established, it must be admitted that the theory of Helmholtz is gradually becoming more and more generally accepted. It is known that the breadth of the basilar membrane increases from base to apex of the cochlea, and Helmholtz assumed that high notes were perceived in the lower portion, whilst low notes were recognised in the upper part. It must be admitted that post-mortem examinations made upon cases in which the hearing power had been carefully tested during life have gone far to corroborate this view, and to place it on a somewhat higher level than that of a merely working hypothesis. Moos, Bezold, Schwabach, Habermann, and Brühl may be named as having contributed largely to this result. It is due to the laborious work of these authors that we are now in a position to diagnose, with some degree of accuracy, affections of the cochlea. Thus, Brühl has recently recorded cases examined carefully during life, and in them the post-mortem appearances of the cochlea and cochlear nerve confirmed the diagnosis. The next question we have to consider is in what respects deafness, due to interference with the nervous apparatus, differs from that caused by changes in the middle ear or meatus. It is sometimes possible to suspect the existence of nerve-deafness in a patient if it be found that the tick of a watch is heard relatively worse than the voice. Certain letters, too, are less readily perceived by the sufferer from nerve-deafness, but a satisfactory conclusion can only be arrived at by a much more elaborate system of testing. As this matter has already been discussed in a preceding article (p. 355), I shall content myself by recalling the main points. In a general way it may be said that Weber's experiment is of a limited degree of value. A medium tuning-fork is placed upon the middle line of the head. If it be heard more distinctly in the better hearing ear this result is suggestive of implication of the cochlea. A modification of this experiment was proposed by Schwabach, who noted the duration of hearing the tuning-fork by bone conduction, and compared it with that obtained on testing a normal ear. This method gives somewhat more accurate results than that of Weber, as by its means the amount of bone conduction can be to some extent measured. Rinne's experiment depends upon the rule that in a normal ear the tuning-fork will be heard longer when held before the ear (air conduction) than when placed over the mastoid (bone conduction), in which case aurists describe the result as positive; when the converse condition of matters exists, as negative. In a case of deafness, then, a positive result suggests that the internal ear is affected. It will be seen that the methods referred to are for the comparison of air conduction with bone conduction; and, so far as our present knowledge goes, we are entitled to conclude that in a deaf patient in whom there is marked preponderance in favour of air conduction, we are justified in suspecting that the impairment of hearing depends upon an affection of the auditory nerve apparatus (centre, trunk, or labyrinth).

Gellé suggested a somewhat different form of test. He found that if a vibrating fork be placed on the bones of the head, and the air-pressure on the tympanic membrane be increased, the sound of the fork becomes less perceived. In cases of labyrinthine deafness the same occurs, but if the obstruction be in the middle ear this will not occur because the phenomenon depends upon the possibility of increasing intra-labyrinthine tension, through pressure communicated by way of the tympanic membrane and ossicles. Bing has observed that in the normal ear a tuning-fork applied to the mastoid when the sound is just dying out, is perceived again if the ear be closed with the finger. This does not occur in cases of deafness due to interference with the sound-conducting apparatus (meatus and middle ear), but does when the labyrinth is at fault.

Having discussed the various characteristics of labyrinthine deafness as tested by bone conduction, let us now consider air conduction. Whilst in middle-ear disease it is usually the low notes which are lost, in affections of the cochlea the impairment is generally, if not always, for high tones. Bezold is dogmatic on this point: "When our deepest C_2(16 V) tuning-fork is heard, we are entitled to exclude an affection of the middle ear." Conversely, this author has found that in uncomplicated labyrinthine deafness this low-pitched fork is heard even when great difficulty is experienced in understanding speech. He has arranged an elaborate tone series composed of tuning-forks, organ-pipes, and a Galton's whistle; but for clinical purposes we may be content with a very low-pitched fork, a medium fork, and a Galton's whistle. Indeed, the more elaborate method has been used chiefly in the examination of deaf-mutes, in order to detect how much hearing remains.

As we have seen, the cochlea shews loss of function by causing deafness—a deafness which differs from that produced in middle-ear disease. Were all cases quite uncomplicated, their differentiation would be easy; but unfortunately we not unfrequently meet with mixed conditions, and in such instances our various tests may give contradictory results. The observer is then thrown back upon information derived from other sources, such as the history, the appearances of the membrane, and so forth. Associated with a degree of loss of function we not unfrequently have evidence of irritation of the cochlea. This shews itself by the production of tinnitus or subjective sensations of sounds. The descriptions of these given by patients vary a good deal, and such descriptions are no doubt largely influenced by education, habitual surroundings, and temperament. Hissing, whistling, rushing, chirping, and humming may be complained of. It is questionable whether the characteristics of the subjective sensations, as detailed by patients, are of any diagnostic value. It has been suggested that direct stimulation of the cochlea gives rise to a high-pitched note, but this is doubtful. Patients who complain of hearing voices and melodies probably suffer from cortical irritation, which may, however, be excited reflexly through the auditory nerve. Whilst disease of the cochlea may give rise to tinnitus, it must be remembered that this symptom is excessively common in affections of the sound-conducting apparatus—more especially in otosclerosis.

According to modern views the semicircular canals have little, if anything, to do with audition. They are assumed to be of the nature of peripheral organs concerned in equilibration. When, therefore, they are attacked by pathological processes vertigo is a prominent symptom.

In investigating the history of a patient, it is important to ascertain whether giddiness—if it be said to have occurred—is of the true rotatory variety. Many persons use the term very loosely. When this symptom is marked, and arises from irritation of the semicircular canals, a sense of rotation is usually present—referred sometimes to the patient and sometimes to surrounding objects. When vertigo of this type occurs the two most probable causes are (1) the labyrinth; (2) intracranial disease, often cerebellar. Collateral circumstances will usually enable the observer to arrive at a correct conclusion. Not uncommonly the paroxysm of giddiness, if it be severe, is associated with or followed by nausea and vomiting. There is usually marked pallor, and the skin is apt to break out in a cold sweat. According to Frankl-Hochwart there may be congestion of the face, whilst the pulse may be at first slow and afterwards quick. He also points out that a true rotatory vertigo may occur in arteriosclerosis, hysteria, and epilepsy, as well as in intracranial disease. He thinks, too, that in epileptics with ear lesions mixed attacks occur. Short of actual occurrence of rotatory vertigo interference with the semicircular canals may give rise to disturbance of equilibration, which require for their detection further investigation. Thus, sometimes, we find that sudden movement of the head, for example turning, will induce giddiness, or the patient mentions this in giving the history. Again, we may only detect imperfections in the balancing faculty by careful testing, and von Stein has done pioneer work in this direction; his methods are, however, laborious, and probably for most cases it will be sufficient to study the patient in (1) standing with the feet together, (2) on one foot, (3) walking and making him repeat these with the eyes shut. If opportunity offer, too, it will be advisable to find out how he can balance himself on a sloping plank, and to try if he can hop backwards in a straight line with the eyes shut. In a case in which complete destruction of the labyrinths is suspected, confirmation may be derived from the fact that in such a case neither rotatory nor galvanic vertigo can be induced.

Another common symptom resulting from irritation of the semicircular canals is nystagmus. Wittmaack concisely expresses the conclusions of main importance in the following words: "The nystagmus is so far characteristic in that it increases on looking to the side, or sometimes only occurs then, and usually sets in more strongly on looking towards the healthy than towards the diseased side." Bárány has found that syringing the ear with water above the body temperature produces nystagmus towards the ear, but that if water below the body temperature be used, the nystagmus is in the opposite direction, whilst water of exactly body temperature causes no ocular movements. He therefore advocates this experiment as a test for the activity of the labyrinth, through which he assumes that this symptom is produced.

It must be remembered that disturbance of equilibration, when due to ear disease, may arise not only from primary changes in the auditory nerve or labyrinth, but may be caused by morbid conditions in the middle or even external ear. Thus, giddiness may be caused by changes of tension within the tympanum, and may even be due to the pressure of impacted wax upon the drum membrane. By physicians and medical men, other than specialists, the term Menière's disease is still much employed. As our knowledge has increased it has, however, become evident that the retention of this term is liable to lead to confusion. It is perfectly true that we may, in an otherwise healthy person, have a sudden attack characterised by symptoms similar to those described by Menière; but it must be remembered that any lesion implicating the semicircular canals, and probably most pathological changes affecting the auditory nerve and the centre representing its vestibular portion, may act likewise. It is, therefore, better to speak of Menière's symptoms, including under this term deafness and giddiness associated in severe cases with tinnitus, vomiting, a tendency to faintness, vasomotor changes (flushing, pallor, or cold sweats), and nystagmus. In very rare instances there may be loss of consciousness, but this is so uncommon that when it is said to have occurred the question must always arise whether syncope or epilepsy may not have been the actual cause. It must be remembered that we may meet with varying degrees of disturbance of equilibration. In bad cases, whilst paroxysms, as described by Menière, may occur from time to time, more or less unsteadiness of gait persists during the intervals. These paroxysms, however, differ in both severity and frequency—the intervals between them varying from days to years. Sometimes, even when the patients notice nothing between the attacks, careful testing will shew the presence of defective equilibration. As stated at the beginning of this section a pathological classification is in the present state of our knowledge unattainable. It is, as before mentioned, probable that very much the same symptoms may be produced by implication of the auditory nerve at its centre, in its course, and terminations in the labyrinth. Where, however, nerve-deafness occurs without vertigo, we may assume implication of the cochlea and its nerve. It is also possible that in some rare cases Menière's symptoms may be due to changes in the semicircular canals without any appreciable implication of the cochlea—producing then little or no deafness. For clinical purposes, however, we may assume that whilst occasionally we meet with nerve-deafness unassociated with vertigo and other vestibular symptoms the converse is so uncommon as to be negligible.

Perhaps for the purposes of this article the best classification we can adopt is as follows:—

(1) Primary disorders of the labyrinth and nerve.
(2) Affections of the labyrinth and nerve following ear disease.
(3) Affections of the labyrinth and nerve due to general diseases.
(4) Affections of the labyrinth and nerve due to toxic influences.
(5) Affections of the labyrinth and nerve due to trauma.

Primary Disorders of the Labyrinth and Nerve.—*Congenital defects of the labyrinth and auditory nerve* have been frequently met with. They may be associated with deficient development of other parts, and lead to deaf-mutism if bilateral. In *advanced* age hearing usually becomes somewhat impaired, although in many cases this can only be detected by careful testing. Defective hearing for high notes is characteristic of *presbyacusis* as it has been designated, but neither tinnitus nor vertigo are complained of.

Neuritis may attack the auditory nerve after exposure to cold. Sometimes the facial is also affected, and occasionally the fifth nerve as well. Deafness due to neuritis is associated with tinnitus, and Menière's symptoms may or may not be present. Wittmaack has recorded a case of nerve deafness without vertigo, in which the autopsy shewed a degenerative neuritis confined to the cochlear nerve. As treatment, counter-irritation over the mastoid, pilocarpine $\frac{1}{8}$th to $\frac{1}{6}$th gr. hypodermically, or $\frac{1}{4}$ gr. by the mouth, attention to the bowels, iodide of potassium or sodium (in large doses if a specific taint be present), confinement to a warm atmosphere, and if giddiness be marked, bromides should be employed.

Primary apoplectiform affection of the labyrinth or Menière's disease was so named because attention was first called to it by the author whose name it bears. Curiously enough, the case which originated the designation was probably not merely labyrinthine, although the only lesion described in connexion with it was a red plastic exudation into the semicircular canals. It is now recognised that such a lesion alone is not capable of causing death, and as Menière's patient, a young girl who, after exposure, was attacked by deafness, intense giddiness and vomiting, died on the fifth day of illness, it is more than probable that some other agency, such as cerebrospinal meningitis, was the real cause of the fatal issue. The symptoms and course of the affection, as we now understand it, are as follows: A person who has hitherto enjoyed excellent health is suddenly the victim of Menière's symptoms—deafness, usually extreme and unilateral, tinnitus, vertigo, vomiting, cold perspiration, flushing or pallor of the face, and nystagmus. The deafness has the characteristics suggestive of involvement of the labyrinth; if any hearing remains that for lower notes only is retained, while bone conduction is diminished or absent. The tympanic membrane is unchanged, and no improvement results from the employment of the Eustachian catheter. The vertigo and other symptoms vary in severity and duration, but the hearing usually remains much impaired or is completely lost. Commonly after a longer or shorter time the power of equilibration is completely recovered, although it is impossible to be certain in any given case that there may not be a return, perhaps after years. Hence it is always well to warn patients as to this possibility, and to advise them, so far as may be, to avoid localities in which the occurrence of such an attack would be specially dangerous.

The treatment of this affection in the acute stage is very much guided by general principles—clearing out the bowels, counter-irritation, and restricted diet. Quinine in large doses has been recommended, but in view of the toxic effects of this drug upon the ear, its administration must be considered of doubtful propriety. In this view I am supported by Frankl-Hochwart, who writes: "I have now eliminated this remedy entirely from my methods of treatment." This author, however, recommends change to a moderately high altitude as very beneficial after the acute symptoms have moderated. In the treatment of vertigo I do not think any other drug equals the bromides, given in fairly large doses, and they act beneficially on the tinnitus as well. When convalescence has been fairly established, and, indeed, for long afterwards, the patient should carefully study his diet and general health. According to my experience, exacerbations of giddiness are very liable to follow any indiscretions. In the treatment of Menière's disease it is always well to begin pilocarpine as early as the general symptoms permit, *i.e.* usually within twenty-four hours. Hypodermic injections of this drug should be made daily—unless contra-indicated by the general condition or idiosyncrasy—for a fortnight. As Politzer pointed out, pilocarpine gives the best chance of success, and sometimes by its means the hearing is saved.

Affections of the Labyrinth following Ear Disease.—*In suppuration of the middle ear* the labyrinth may become involved; as this subject has been fully dealt with in another section (chronic middle-ear suppuration), a few words only will be here required.

In *scarlatina*, and sometimes in *measles*, evidences of labyrinthine complication may shew themselves even in the early stages of the acute middle-ear processes which so often accompany these diseases. The deafness then usually becomes almost absolute, and if it be not complete, will have the characteristics already insisted upon, whilst giddiness may or may not be a prominent symptom. In addition to appropriate treatment directed to the middle ear, pilocarpine should then be immediately employed. In *chronic middle-ear suppuration* the labyrinth may become infected, and such infection may be followed by caries and even exfoliation of parts of the labyrinth.

In chronic middle-ear catarrh, but more particularly in *otosclerosis*, as the disease progresses the labyrinth may become obviously affected. Deafness then becomes almost complete, the previously retained perception of high notes is lost, and hearing by bone conduction is reduced to a minimum or vanishes. In these cases, too, inflation often makes the hearing—when still sufficiently retained to make testing possible—worse. Unfortunately no treatment is of much avail, although hydrobromic acid may be prescribed if tinnitus be much complained of, and strychnine is sometimes useful as a tonic.

A form of *progressive nerve-deafness* has been described, occurring usually in old people, and probably differing from presbyacusis in degree only. Alexander has recorded the results of examination of the labyrinth in a woman who was completely deaf in the left ear and partially so in the right. Whereas on the left side he found complete destruction of the organ of Corti with a slight degree of atrophy of the cochlear nerve and spiral ganglion, on the right there was only partial destruction of the hair-cells, but no nerve degeneration. Thus it was shewn that the atrophy of the cochlea was primary. Manasse has described a number of examinations in which degenerative changes were found in the organ of Corti, spiral ganglion, and auditory nerve. Clinically the records were rather defective; deafness was progressive, the membranes were normal, the voice badly heard, the lower tone-limit normal, the upper tone-limit lowered, bone conduction shortened, and Rinne's experiment positive. He was unable to say anything definite about disturbance of equilibrium.

Affections of the Labyrinth associated with General Diseases.—*Meningitis*, whether acute or chronic, may implicate the auditory nerve and internal ear. *Epidemic cerebrospinal meningitis* is, however,—in countries in which it is epidemic—a very fruitful source of deafness. This disease may cause changes either in the nucleus of the auditory nerve, in its course, or in the labyrinth. Deafness seems to occur most frequently in the first and second weeks of illness. After recovery the hearing is often completely lost on both sides, and on first getting up marked disturbance of equilibration is noticeable, the patient, if a child, often having to learn to walk again. It has been found that the giddiness gradually passes off, but that the deafness persists. During epidemics an *abortive form of cerebrospinal fever* frequently occurs. The patient, usually a child, complains of headache, becomes feverish, the head may be retracted, there is sickness, flushing, delirium, perhaps convulsions, and even coma may supervene. When recovery takes place, as it usually does, in a few days, the patient is found to be deaf and giddy—the former symptoms persisting and the latter passing gradually off. It is important to bear in mind that even here, where cerebrospinal meningitis is rare, cases answering to the above description are not uncommon in hospital practice.

Mumps may produce labyrinthine deafness, which is fortunately usually confined to one ear. The loss of hearing commonly occurs at the end of the disease. Whilst sometimes vertigo and tinnitus are also complained of, these symptoms are more commonly absent.

Influenza, diphtheria, pneumonia, enteric, small-pox, and whooping-cough may be followed by deafness having the characteristics of the labyrinthine form. The same is true of *osteomyelitis, scarlatina, measles, and pulmonary tuberculosis.* Siebenmann has endeavoured to elaborate a clinical picture of the deafness due to the first-named affection. The osteomyelitis he refers to is the form which occurs in young people. The patients are attacked by fever and perhaps delirium, which may precede the appearance of bone inflammation by days or even weeks. Sometimes only one long bone is affected, sometimes multiple areas are involved. The ear symptoms usually appear after the disease has run its course. In more than half the cases deafness is ushered in by tinnitus, and, later, giddiness and vomiting occur. Sometimes hearing is lost in a few hours or days, but the process may be gradual. In most cases bilateral deafness due to labyrinthine changes follows. In measles and scarlet fever, as we have seen, there may be involvement of the labyrinth secondary to middle-ear suppuration, but Bezold states that in the latter there may occur a form which in his opinion depends upon neuritis of the auditory nerve. The same author has also described a *nerve-deafness* in *pulmonary tuberculosis.* It usually comes on rapidly, and has been found to depend upon neuritis.

In *leukaemia* sudden deafness may occur, associated with the chain of symptoms which bears the name of Menière; occasionally impairment of hearing comes on more gradually. Post-mortem examination of cases has shewn extravasations of blood in the labyrinth, and not unfrequently organised inflammatory products have also been found. Sudden bleeding into the internal ear may occur in *Bright's disease*, in *pernicious* and probably also in *simple anaemia.* The occurrence of acute deafness depending upon affection of the nerve has also been observed in *diabetes*, but no pathological data on this point exist.

Of all chronic diseases, that which most frequently attacks the internal ear is *syphilis*. In *acquired syphilis* the ear affection may occur at almost any stage of the disease. I have known a patient attacked by deafness within a very short period of primary infection; in other cases it may come on after a lapse of years. It has been noted that syphilitic disease of the labyrinth is much more common in men than in women. In cases in which post-mortem examination has been possible small-celled infiltration, changes denoting periostitis, and atrophy of ganglion cells have been found within the labyrinth. Fortunately in a considerable number of instances, only one ear is attacked, and when this is so—according to my experience—there is no great risk of subsequent affection of the other. Acquired syphilis of the internal ear may assume either of two clinical forms. (1) In a certain proportion of cases the patient is suddenly attacked by deafness, tinnitus, giddiness, and the other symptoms known as Menière's, and only the history gives a clue to the cause. (2) The patient may, however,—and in my experience this is the more common form—become suddenly deaf and suffer from tinnitus without any disturbance of equilibration. In such cases we may assume that pathological changes are confined to the cochlea, and the affection has been described by Roosa as cochlitis. I should feel inclined to suspect the existence of syphilis in any case in which sudden deafness, having the characters of the labyrinthine form, occurred without accompanying disturbance of equilibration.

In *hereditary syphilis* labyrinthine deafness is of frequent occurrence. It usually shews itself at a later period than keratitis, and is perhaps most common between the ages of ten and fifteen. According to Bezold, however, its onset may be delayed until many years later. It is important to bear in mind that the victims of hereditary syphilis of the internal ear almost invariably suffer from evidences of Eustachian obstruction. The tympanic membranes are often very much indrawn, and a casual observer, who dispensed with the precaution of investigating the history and testing the hearing, might readily assume that he had to deal with a case of middle-ear catarrh. Investigation will, however, shew that the deafness has progressed much too rapidly, and reached a degree rarely due to this cause alone. The patient will usually be found to have evidences of interstitial keratitis, and examination of the mouth will shew the characteristic notched incisors. Finally the character of the deafness will correspond with that found in affections of the inner ear, and usually no improvement follows inflation. Perhaps it is hardly fair to pass from this subject without stating that sometimes the presence of middle-ear changes rather interferes with obtaining typical results with hearing tests, and again it is quite possible that a degree of improvement may follow the use of the catheter or Politzer's bag. Commonly, however, the history, course, and presence of other phenomena (keratitis and teeth) will lead to a correct diagnosis.

Syphilitic meningitis may implicate the trunk of the auditory nerve, and in such cases the deafness is usually associated with facial paralysis. Frequently, too, other cranial nerves may be involved. *Locomotor ataxia*—which is probably in most cases of syphilitic origin—often causes gradual nerve-deafness, which depends upon degenerative changes in the nerve trunk.

The treatment of syphilitic deafness generally is by no means hopeful. Unlike other lesions produced by the same cause, little benefit usually accrues from either iodide of potassium or mercury. Still both remedies should be employed carefully and thoroughly in every case. In addition, however, pilocarpine must be administered by hypodermic injection as soon as the case is diagnosed. Sometimes, if the patient be seen early, this drug may save the hearing—more particularly in acquired cases in which Menière's symptoms set in acutely.

Labyrinthine deafness may be met with as a result of an *epileptic fit*. Urbantschitsch has described a curious case in which the patient became completely deaf after a fit, remained so for two years, and then recovered his hearing after another epileptic seizure.

A form of deafness has been described as *angioneurotic*. It is accompanied by pallor of the face, sickness, giddiness, and tinnitus, which pass off in the course of a few minutes. Probably closely allied to this condition is the defective hearing which sometimes results from *extreme emotion*, only in some of these cases complete deafness has remained permanently. As to the pathology of this condition we know nothing.

Pathological changes within the cranium may cause deafness. Probably destruction of one auditory centre will cause only temporary impairment of the opposite ear, but where the lesion is bilateral deafness results. Thus, Wernicke and Friedländer recorded a case in which both temporal lobes were the seats of gummas, and complete bilateral deafness resulted. Bezold describes a "*mid-brain deafness*," and as his description is important, alike to aurist and physician, I shall give a translation of his views and observations. "There is no other part of the brain, disease of which is so frequently accompanied by disturbances of hearing as is the case in tumours of the mid-brain; according to statistics, which with regard to hearing make no claim to exact accuracy, marked deafness is present in 20 per cent of cerebellar tumours, in 25 per cent of tumours involving the pons, and in 34·5 per cent of growths in the mid-brain. As I have been able to shew positively in a large series of cases it is never a lesion of the flat area of the corpora quadrigemina, but only of the fillet region of the tegmentum which causes deafness in such cases. This occurs relatively late in tumours of this area, among which we must class growths of the pineal gland. The first symptoms are headache, defects of vision, excitement, and in most cases a brief apoplectic seizure. Later, changes of speech, disturbances in the movements of the eyeballs, ataxia, epileptic attacks, interference with the movement of the body and limbs, and facial paralysis are added. Of rarer occurrence are incontinence, disturbances of the reflexes, paralysis of deglutition, disturbance of sensibility, polyphagia, and rise of temperature. The deafness is rarely observed at the end of the first month, and usually is not noted until three to six months after the beginning of illness. The rapidity of its onset is of course dependent upon the nature and rapidity of growth of the tumour. Bone conduction is at first shortened and then absent. Hearing power in the cases tested by me with the continual tone series was first lost for low notes, but later for all tones equally in such a way that the auditory field was reduced from above and below so as only to leave a small island." According to Bezold neither tinnitus nor giddiness are much complained of.

Tumours of the pons, situated as some of them are in the angle between this structure and the cerebellum, are apt to produce ear symptoms. To quote Bezold again: "The deafness is usually unilateral, occurring on the side corresponding to the tumour, and this symptom, both as to time of occurrence and intensity, generally becomes prominent. Its unilateral character, as well as the early occurrence of giddiness, nystagmus, facial paralysis, and impairment of deglutition, serve to differentiate these growths from tumours of the corpora quadrigemina in which ophthalmoplegia dominates the clinical picture."

Tumours of the cerebellum also may cause deafness; and Schwartze has recorded an interesting case in which a unilateral cerebellar growth caused bilateral deafness.

Intracranial aneurysms, too, may cause loss of hearing, more particularly when the basilar artery is at fault. In these cases severe pulsating tinnitus, often audible with the stethoscope, is a pronounced symptom.

Of course, most of these conditions are incurable, excepting when they depend upon syphilis, or when they are amenable to surgical treatment.

More or less marked deafness may be due to *hysteria*. In certain cases its diagnosis is not difficult. Thus, if an hysterical patient—usually a woman—becomes suddenly deaf without obvious changes in the middle ear and without the occurrence of giddiness, the suspicion of hysterical deafness will be justified. Further confirmation will be obtained by the presence of anaesthesia, affecting either the whole of one side of the body or certain parts. Thus, in some cases the external meatus and tympanic membrane may lose their sensibility. Sometimes the phenomenon of transfer may be observed, that is, all the symptoms pass from one side to the other. As a rule hysterical deafness is not accompanied by either giddiness or tinnitus. The next question which will occur to the reader is as follows:—Is there any special peculiarity as to hearing tests? According to Gradenigo the deafness will be equal for notes of different pitch, that is to say, there will not be marked difficulty in detecting low notes as in middle-ear deafness, nor high notes as in labyrinthine disease. Weber's experiment may give varying results; the hearing may change rapidly, either spontaneously or after the employment of some remedy. Gradenigo also lays considerable stress upon the presence of diminished excitability of the auditory nerve, for he believes that in cases of severe organic lesions of the nerve or labyrinth electric excitability is increased. Now what has been just stated certainly will facilitate diagnosis, provided obvious stigmata of hysteria are present, and provided too that the deafness is not complete. In such a case as the following these methods fail us, and for this reason I venture to give the history at length. A girl of nineteen consulted me on May 6, 1905, and the following is a verbatim copy of my notes:—"Deaf in right ear for eight weeks; epidemic of mumps at school; patient, however, seems to have had little glandular swelling, was in bed, suffered from neuralgia of right side of face. Both ears were somewhat affected. The left quickly recovered, but the right did not. Right ear has been painful, but is not so now; never discharge; Politzerisation has been used daily. A fortnight ago, when ear was painful, there was swelling and tenderness over mastoid (?). Never giddy. Hearing distance, the watch not heard, very loud voice not heard; indeed the voice used was so loud that it ought to have been heard by the other ear, which was of course closed. L. Watch $\frac{30}{30}$, very low whisper at 18 feet. Tuning-fork only in left ear from middle line; it was also heard when the left ear was closed. Galton's whistle was not heard with right ear. The right tympanic membrane was a little thickened, but moved freely on Valsalva's experiment. Menstruation said to be regular. Careful testing (partly by me and partly by her own medical man, who kindly reported to me) revealed no anaesthetic areas anywhere. Two years ago was a little hysterical; mother hysterical. Diagnosis—mumps or hysteria. Treatment—general; rest, arsenic and iron. *June* 26.—Hearing on right side normal; in the beginning of June heard rushing sound in right ear but still did not hear. On the second occasion that this occurred heard at once for a short time but hearing went away. It then began to come back frequently, but she still has occasional periods of not hearing. Since the last visit, however, the deafness has remained away." In such a case certainty could only be attained after the hearing had returned, because exactly the same ear symptoms might have resulted from mumps, although the inability to hear a loud voice with the good ear closed was suggestive.

In a general way I am inclined to rely upon the following points in the diagnosis of hysteria:—(1) Is there marked discrepancy between the history and the results of objective examination? (2) Do the history and manner of the patient suggest hysteria? (3) Do history and results of examination accord with any of the recognised forms of organic deafness? (4) Is there any evidence of sudden improvement of hearing when the patient is interested? (5) Is there any anomaly of hearing; for example, does the patient with the good ear closed fail to hear a voice so loud that it ought to be detected with the closed ear? Does he or she hear the tuning-fork by bone conduction worse when the good ear is closed? Do hearing tests give varying results?

Deafness due to Toxic Causes.—Of drugs acting upon the ear *quinine* is the one whose symptoms are most generally known. Large doses cause not only deafness, having the characteristics of the labyrinthine form, but vertigo and tinnitus also. As a rule these phenomena pass off rapidly, but in view of recent evidence and clinical experience it is questionable if a chronic nerve-deafness may not be a result of large and frequent doses of quinine. Of course the difficulty in arriving at a definite conclusion is that the persons who have taken such large doses have usually also suffered from malaria, and chronic nerve-deafness is often met with in persons who have spent long periods in malarial districts. At one time it was thought that quinine produced hyperaemia of the labyrinth with haemorrhages; but as I pointed out long ago, this effect was improbable, because in quinine amblyopia the disc has always been found anaemic. Panse has now shewn that the congestion and haemorrhage are due to suffocation or to death-agony. Moreover, Wittmaack has proved that quinine poisoning produces various changes in the cells of the spiral ganglion, and the same observer did not find any haemorrhages in the tympanum, labyrinth, or auditory nerve.

Salicylate of sodium gives rise to exactly the same labyrinthine symptoms as quinine, and Blau has shewn that similar changes in the ganglion cells are produced.

The effects of *tobacco* and *alcohol* upon hearing have not yet been sufficiently studied. Mr. Wyatt Wingrave has described seventeen cases of deafness due to excessive smoking; of these approximately four-fifths were improved by abstinence, tonics, and bromides, but only three were quite cured. Alt has also described similar cases in which giving up smoking effected a cure. Bezold is inclined to ascribe more importance to alcohol than to tobacco. He observed rapid deafness in three cases, and in two of them abstinence was followed by a return of hearing power.

Of agents other than those mentioned, *mercury, lead, phosphorus, hasheesh, chenopodium, carbon monoxide, chloroform, and ether* have been mentioned as occasional causes of deafness, probably usually temporary.

Traumatic Affections of the Auditory Nerve and Labyrinth.—*Direct lesions of the labyrinth* are somewhat rare. They may be caused from a sharp instrument, such as a knitting-needle being forced into the ear and penetrating the vestibule, usually, if not always, through the fenestra ovalis. Again, forcible attempts to extract a foreign body may produce a lesion of the internal ear. Such injuries are immediately followed by intense giddiness, vomiting, and tinnitus, which gradually pass off. Complete deafness, however, usually remains. Not uncommonly the facial nerve is also injured, and then, of course, facial paralysis will be a prominent symptom. Only in a proportion of cases has a flow of cerebrospinal fluid resulted from direct injury to the labyrinth. It must, however, be understood that acute inflammation of the middle ear is apt to occur, and its severity will depend upon the nature of the instrument and other factors. Treatment must be chiefly based on instructions given in a previous portion of this work for dealing with injuries of the drum-membrane, while rest, non-stimulating diet, attention to the bowels, and the treatment of the most prominent symptoms by bromides or other suitable remedies will be called for.

Labyrinthine deafness following blows or falls on the head is often due to irreparable lesions of the auditory nerve or labyrinth, although in some cases loss of hearing is due merely to concussion of the auditory apparatus,—whether centre, nerve, or labyrinth is often not easy to determine in any given case. In fracture of the base of the skull, deafness in one or both ears is often observed after the patient has recovered sufficiently to notice impairment or loss of hearing. The specialist has seldom an opportunity of examining such cases until a considerable interval has elapsed. The usual history is somewhat as follows: After a blow or fall the patient became unconscious; blood and sometimes watery discharge may have been noticed to have come from one or both ears, in which case it may be assumed that there had also in all probability been a rupture of the drum membrane. After treatment for a varying period, the patient so far recovered as to take note of his surroundings. He then noticed that he was deaf in one or both ears, and commonly such deafness is associated with giddiness and tinnitus, and occasionally facial paralysis is also present. The subsequent history of these cases is usually unfavourable. The giddiness invariably disappears after a longer or shorter period, the tinnitus may or may not persist, but commonly the hearing remains permanently impaired or lost. The prognosis depends upon the amount of deafness and its duration. Even if the patient be quite deaf immediately after the accident, hearing may eventually be recovered; but if he remains so for some weeks the prospects are most unfavourable. It is doubtful whether any treatment has much effect in restoring function.

A blow on the ear may cause deafness, either by injuring the drum membrane or causing concussion of the labyrinth. In many cases both lesions are present. The same results may follow *violent explosions, e.g.* gunpowder or gas. Very sudden exposure to a *loud sound* may also cause deafness. The diagnosis depends of course upon the result of hearing tests as well as upon the history. The symptoms vary according to the extent of the lesion. When the concussion of the labyrinth has been severe the patient feels giddy or may even faint. Tinnitus is usually experienced immediately, and soon afterwards he realises that he is deaf in the injured ear—a deafness which tests shew to be labyrinthine in character. Hearing may be completely lost or only impaired. Diagnosis is relatively simple if the middle ear be intact, but if the membrane be also injured very careful examination will be required in order to assign to each factor its proper importance. The presence or absence of bone conduction, and the power to hear high or low notes, must be carefully investigated. As a general rule, if hearing be not completely lost, improvement will gradually take place, but we cannot tell how far this process of repair will go. If the deafness be absolute, some amount of hearing power may eventually be recovered, but this cannot be promised.

Constant exposure to loud sounds is a frequent cause of deafness. This condition is perhaps most commonly found in boilermakers—hence the generic term "*Boilermaker's deafness.*" Coopers, tinsmiths, locksmiths, carpenters, factory-workers, engine-drivers, and firemen also suffer in the same way. In a certain number of these cases tinnitus is complained of, but frequently the only symptom is gradually increasing deafness. In not a few instances examination of the membrane gives indications of middle-ear catarrh; indeed, so often have I found this to be the case that I cannot but think that middle-ear changes—more particularly Eustachian obstruction—act as a predisposing cause. The deafness, however, is usually of the labyrinthine type, with lowering of the upper tone-limit and retained hearing of low notes. Habermann and Brühl have made histological examinations of the ears of patients who were affected by this form of deafness: the former found atrophy of the organ of Corti, which in most cases affected only the lower parts of the cochlea, while the latter detected atrophy of the spiral ganglion and its nerves passing to the first turn of the cochlea, together with defect of Corti's organ in the same area. Of course the only treatment which can be prescribed is prophylactic, and if the patient cannot give up his work, he ought to wear obturators in his ears, either of rubber or of cotton wool saturated with vaseline.

Injuries of the auditory nerve apparatus have been described, in which the cause has been *lightning*, either striking the patient or conducted to his ear by the use of a telephone during a thunderstorm.

Caisson workers are liable to nerve-deafness if too sudden decompression is allowed to take place. The symptoms usually come on acutely, and include giddiness and vomiting as well as tinnitus and deafness. The hearing may be recovered after some days, or may remain permanently lost or injured. One or both ears may be affected. In these cases the symptoms are probably due to air embolism affecting the labyrinth or auditory nerve. Bezold suggests that probably the best treatment would be immediate return to the compressed-air chamber.

Artillery-men and *sailors on board men-of-war* may suffer from nerve-deafness, which is of the type described as occurring in boilermakers. *Sportsmen*, whether with gun or rifle, are liable to the same effect. In the cases in which deafness results from shooting small game, it is, owing to the position of the head adopted by most gunners, the left ear which suffers rather than the right. Sometimes the patients also complain of headache after a long day. In such instances, this form of sport must be interdicted, or if persisted in, plugs must be worn on both sides. As the ears of persons who suffer from Eustachian obstruction are more liable to be affected than others, steps must be taken to remedy this defect if present.

In view of the tendency of recent legislation it is probable that the question of *simulated deafness* will in the near future become more important to British aurists than heretofore. On the Continent the matter has always attracted much attention, owing to the attempts of recruits to avoid military service. Complete bilateral deafness is rarely successfully simulated, because of the initial difficulties involved. Supposing, however, that these have been got over, and that the patient acts his part well, detection may be extremely difficult. It has been proposed to watch the face while an assistant makes disparaging remarks behind the patient's back; again, the sudden calling of his name during sleep may give a result which justifies suspicion. In using this test, however, care must be taken that no vibration is produced, and that the breath does not impinge upon the patient's face. As an extreme measure, chloroform may be used, and questions asked while the suspected person is still partially under the influence of the drug. This, however, presupposes assent, which is quite likely to be withheld. Absolutely deaf persons often lose the power of modulating their voices, and where this symptom is present it affords evidence of *bona fides*. Unilateral deafness is more commonly simulated, and in a sense more readily detected. In such cases it is well to make the patient repeat the history on several occasions with a view of noting any discrepancies. The hearing is then tested repeatedly in the ordinary way while the patient is blindfolded. Any marked variations in the results will give grounds for suspicion. While making these tests the auricles should be closely watched. In many persons, when listening intently, they are seen to move, and when this movement is detected it affords, in my opinion, a strong presumption of honesty. The next test depends upon the fact that, given a good ear even if this be stopped up, moderately loud conversation in its immediate neighbourhood will be heard. The malingerer in these circumstances will often state that he hears nothing. A vibrating tuning-fork of medium pitch is then applied to the middle line of the head, and the suspect will probably state that he hears it in the supposed good ear. If this be now occluded he will quite probably give his case away by denying that he hears it at all. An ingenious use of the binaural stethoscope has been proposed. One of the ear branches is tightly closed with a cork, and the corresponding ear-piece is inserted into the patient's good ear. Words are then whispered into the instrument, and if they be repeated our suspicions should be aroused. Care must, however, be taken that the words are not spoken sufficiently loud to be perceived by the good ear through air conduction, and the observer must stand behind the patient. If after these tests have been employed doubt still exists, the ears of the suspect must each be furnished with long conversation tubes. Into each of these two assistants whisper words which the patient is asked to repeat. After this has gone on for a time even the most accomplished malingerer will betray himself by repeating what has been spoken into the deaf ear. This experiment may be elaborated by having the tubes conducted into another room.

Even when the aurist has arrived at the conclusion that the patient is really deaf his difficulties are by no means over, for it is often a most difficult if not impossible task to decide whether the deafness has really dated from the injury which is supposed to have caused it. In many cases the conscientious expert cannot commit himself further than to state that the results of his examination are consistent with such a proposition. After this we are dependent upon the evidence of others as to whether the impairment or loss of hearing immediately followed the alleged cause, or whether it existed before. Employers of labour would, therefore, be well advised to have the hearing of proposed employees tested before engaging their services. P. M'Bride.

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P. M.

DEAF-MUTISM

By James Kerr Love, M.D.

Definition.—Deaf-mutism is an absence of hearing sufficient to prevent the acquisition of speech by the deaf child, or a loss of hearing sufficient to endanger the already acquired speech of the child. This degree of absence, or loss of hearing, is not a uniform or absolute quantity, but for convenience it has been called by the writer "surdism."

Etiology and Pathology.—Deaf-mutism is either congenital or acquired. This classification is convenient rather than exact, the diseased condition causing the deafness in the two classes of cases being much more alike than appears on the surface or than is generally supposed.

The number of deaf-mutes in Europe is about 1 to 1350 of the population. In Switzerland it is as high as 1 to 408, in Austria 1 to

765, in Sweden 1 to 977, and in Germany about 1 to 1000 of the population. In many countries, on the other hand, the deaf-mute rate is below the average of 1 to 1350. In Ireland it is 1 to 1398, in Denmark 1 to 1500, in France 1 to 1600, in Scotland 1 to 1860, in Italy 1 to 1860, in England 1 to 2043, in Belgium 1 to 2250, and in Holland 1 to about 3000 of the population. In mountainous countries, where the population is sparse and the food of the people is poor, consanguineous marriages are common, the conditions favouring degeneracy are present, and the congenital deaf-mute rate is high. Flatter countries are more populous, the standard of living is higher, intercommunication is easier, and, as a result, consanguineous marriages are less common and the congenital deaf-mute rate is lower. On the other hand, the epidemic diseases—measles, scarlet fever, and meningitis—ravage densely populated countries more than sparsely populated countries, and the acquired deaf-mute rate is greater in the former. Generally speaking, the deaf born are slightly in excess of those whose deafness has been acquired; but from year to year the ratio varies with the occurrence of the epidemic diseases chiefly responsible for acquired deafness.

Congenital deafness is hereditary. It may appear in the direct line, or the offspring may be deaf from reversion to the characters of an earlier progenitor. Deafness in the collateral branches of a family may be the only discernible trace of the hereditary tendency. Congenital deafness sometimes occurs in association with malformation of other parts. Malformation of the auricle is rare amongst deaf-mutes. When it occurs it may be associated with malformation of the deeper parts of the ear, or these latter may be normal. Consanguinity in parents emphasises family features in the offspring. Family defects and tendencies to disease are apt to become accentuated in this way. Consanguineous marriages produce deteriorated offspring, and deaf-mutism is much commoner after such marriages than after marriages in which the parents are not related. Amongst animals when perfect specimens only or chiefly are used, inbreeding may be carried on to a very large extent without visible bad effect. In the human race, in which every family has some transmissible taint, consanguineous marriages are often immediately followed by deterioration, of which deaf-mutism is an example.

Acquired deafness causing mutism is due in the majority of cases to one of three diseases—meningitis, scarlet fever, or measles. The above is the order of frequency, and these three diseases account for nearly two-thirds of all cases of acquired deaf-mutism. Other comparatively frequent causes are enteric and other fevers, whooping-cough, and diphtheria. Congenital syphilis would seem, from an examination of the eyes of deaf-mutes, to be a frequent cause of both congenital and acquired deaf-mutism. Middle-ear suppuration, apart from scarlet fever and measles, hardly ever causes such severe deafness that dumbness is the result. Post-nasal adenoids alone never cause deaf-mutism.

Morbid Anatomy.—Many, especially of the earlier, post-mortem examinations of the ears of deaf-mutes are incomplete, and in many the microscope has not been used. The number in which there is a clear clinical history and a careful examination of all sections of the ear and of the auditory nerve and brain is still small. Such examinations as have been made shew that acquired deaf-mutism is almost always due to disease of the internal ear,—disease which, after destroying or greatly altering the tympanum and its contents, has spread most commonly by way of the foramen rotundum, and has caused inflammatory changes involving destruction of the membranous labyrinth and of the nerve structures which it supports. In a smaller number of cases the danger approaches from the side of the brain, and it is possible that some cases of deaf-mutism are due to a primary labyrinthitis.

In congenital deafness, the changes on which the deafness depends are not essentially different from those described above, except in the few cases depending on arrest of development and malformation. They consist of obliteration of the normal nervous structures by inflammatory new formations, chiefly osseous. It is not possible to estimate the proportion of cases due to malformation, but it is undoubtedly smaller than was formerly supposed. After a series of years it cannot be decided in many cases whether a given structure has never existed or has been obliterated.

General Characters of Deaf-mutes.—If the deafness have been acquired and have come on before the age of five years, dumbness will follow, unless special efforts be made by a teacher to preserve the speech. Apart from the deafness and its consequence—the dumbness—deaf-mutes do not differ physically from hearing people. If properly cared for either in the home or the institution, deaf children develop in height, weight, and chest measurement much as hearing children do, and when they reach adolescence compare favourably with the latter, except that the head measurement is slightly less in circumference. This defect is due to the fact that during the years of rapid brain growth—from two to seven years—function is much less active in the deaf than in the hearing child, and cranial development is therefore less. Degenerate and weak-minded children are commoner amongst the deaf than amongst the hearing. Nearly all deaf-mutes have some hearing left. Amongst congenitally deaf children it is very rare to find one totally deaf; amongst those whose deafness has been acquired, total deafness is more common, but hearing, when it is present, is usually greater than amongst the congenitally deaf, hence most of the semi-deaf children have been born hearing, and have lost this sense in early years. These semi-deaf children hear, and with some practice distinguish vowels, consonants, and in some cases words and sentences when these are spoken near the ear. When hearing is completely lost by children who have been born hearing, and this has not occurred before the age of five years, some speech remains during life, and the child is called a semi-mute. The hearing of deaf-mute children should not be tested by watches, acoumeters, and such means, as their deafness is too profound for these methods (cf. p. 354). The only two tests of practical value are the human voice and the tuning-fork held opposite the ear. Bone-conduction testing is not of much value and is difficult to apply. The human voice is the most important test for hearing in deaf-mute children. It should be used by the side of or behind the deaf child, else he will lip-read the speaker and the test will be valueless. But the speaker must remember that an untaught deaf-mute cannot imitate or reproduce what he hears. It is better, therefore, to postpone the testing of deaf children till after the first or even the second school-year is past. The voice, or other loud sound, is used to prove the presence or absence of hearing; the tuning-fork is used to ascertain its distribution. The remaining hearing of deaf-mute children nearly always exists in islands, which are often outside the speech area of the scale. When the hearing islands are within the speech area of the scale, and when the hearing is considerable in degree, vowels, consonants, and other expressions of the human voice are distinguished, and the deaf-mute acquires speech more readily, whilst at the same time the intonation of his voice is more natural and pleasanter. (See Table shewing Residual Hearing in Deaf-mutes.)

TABLE shewing the Residual Hearing in Deaf-mutes and the Effects of Residual Hearing and Speech on the Education of Deaf Children.

Case.	Age of Reported Onset.	Assigned Cause.	Deaf Relatives.	REMNANTS OF HEARING.											Condition of M. T.	Hearing of Speech.	Speech used.	Intonation.
							Speech Area.											
				C_{11} 16	C_1 32	C 64	C 128	C^1 256	C^2 512	C^3 1024	C^4 2048	C^5 4096	C^6 8192	C^7 16,384				
I	Born deaf	..	..	Right Le	ear ft ear			a..	...... e^1....g^2	......	...... e^3......a^4		..a^5		Intact	Hears voice	None	..
II	Born deaf	..	2 brothers						e^1....g^2	c^3	e^4.. g^4		..g^5 ..d^5		Intact	No vowels	None	
III	1 year	..	..		D_1..	......	...... B	....	..c^2 f^2 g^2 c^2 h^2						Intact	Nil	Little	Fair
IV	Born deaf	..	3 brothers deaf and dumb				B	...... g^1..	......g^2 ....a^2		c^4 g^4 a^4				Intact	Hears A and O	Reads sentences	Fair
V	Born deaf	..	..						g^2..	..e^3	f^4 g^4 f^4 g^4				Intact	Nil	Fair	Fair
VI	4 years	Fall	..							c^3..g^3 nil	f^4..c^5				Intact	Nil	Reads well	..
VII	Born deaf	..	..	G_2.. G_2..	....D_1	..G ..G			e^2..	...... d^3..a^3	......	......	......	..e^6	Intact	Nil	..	..
VIII	Early	Tubercle R. facial palsy	..		 A_1	G..	....	....	....	....	....	....	..a^5 ..a^5	g^6	Gone	All vowels	Unused	..
IX	Born deaf	..	..						 g^2..	d^3..a^3 ....a^3	 d^4				Normal	Nil	None	..
X	Born deaf		..			G..	...... B	....	....	...... a^3	....a^4 f^4..		 ..a^5		Normal	Hears "A"	Articulates well	Good
XI	4 years	Measles	..			G	......	g^1	..c^2	c^3....	c^4	......	..a^5	d^6..d^7	Gone	No vowels	Semi-mute	Good
XII	1½ years	..	..							c^3...... c^3......	....	....	..a^5 ..a^5		Normal	Distinguish "A" and "O"	Reading good	Good

TABLE shewing the Residual Hearing in Deaf-mutes, etc.—*Continued*

Case.	Age of Reported Onset.	Assigned Cause.	Deaf Relatives.	REMNANTS OF HEARING. Speech Area (C 128 – C^2 512). C_{11} 16, C_1 32, C 64, C 128, C^1 256, C^2 512, C^3 1024, C^4 2048, C^5 4096, C^6 8192, C^7 16,384	Condition of M. T.	Hearing of Speech.	Speech used.	Intonation.
XIII	Born deaf	..	2 deaf-mute brothers	A_1……………a^3 f^4 a^4 G a c^2 c^3	Normal	Hears low voice.	Reads fairly well	Good for low tones.
XIV	2 years	..	..	G……e G……………a^3 g^4……d^5	R. Perfor. L. Normal	Vowels except E	Reading fair	Good
XV	2½ years	Fever	..	a……g^1 A_1……………c^2 e^2……a^3 c^4……………d^6	R. Perfor. L. Chalky	A, O, and P	Distinct	Good
XVI	Born deaf	.	2 others deaf-mutes	g^1……………e^3 c^2 g^2……a^3 b^4……d^5	Intact	Nil	Fairly distinct	Fair
XVII	Born deaf	..	1 cousin deaf-mute	c……a g^4……d^5 G……c e……d^1	Normal	None	Poor	Poor
XVIII	Born deaf	..	..	g……………b^4 a……………a^3 c^4……f^4	Normal	Slight	Fair	High-pitched
XIX	Born deaf	..	2 sisters deaf-mutes	G……………c^2 c……a	Normal	Doubtful	Poor	Poor
XX	10 months	Meningitis	..	g^2……d^3 ……b^4 g^2……c^3 g^3……	L. cicatrix?	None	Poor	Squeaky
XXI	Born deaf	..	Brother and father deaf-mutes	G……g c^3 c^3	Normal.	None	Reading fair	Fair
XXII	Born hearing	..	..	D_1……………d^3 nil	R. Normal: L. Gone Mastoid Op.	None	Poor	Poor
XXIII	3 years	Scarlet fever	..	c^3……………d^5 D……………a^3 g^4……c^5	R. Perfor. L. Scarred	All vowels	Reading good	Very fair

TABLE shewing the Residual Hearing in Deaf-mutes, etc.—*Continued*

Case.	Age of Reported Onset.	Assigned Cause.	Deaf Relatives.		REMNANTS OF HEARING Speech Area. (C 128, C^1 256, C^2 512) C_{11} 16, C_1 32, C 64, C 128, C^1 256, C^2 512, C^3 1024, C^4 2048, C^5 4096, C^6 8192, C^7 16,384	Condition of M. T.	Hearing of Speech.	Speech used.	Intonation
XXIV	Born	..	Sister deaf, cousin imbecile		g^2 $c^3 .. f^3$ $c^3 .. a^3$	Normal	None	Reading fair	Very fair
XXV	5½ years	Scarlet fever	..		Group of cases of acquired deafness in which no residual hearing exists.	Both discharge.	None	Good	Good; semi-mute
XXVI	9 years	Scarlet fever	..			Chronic Otitis Media	None	Distinct	Good; semi-mute
XXVII	9 years	Fall	..			R. Normal L. Retracted and red	None	Good	Good; semi-mute
XXVIII	9 years	..	..			R. much reddened	None	Distinct	Good; semi-mute
XXIX	3 years	Scarlet fever	..			R. two cicatrices L. gone	None	Moderate	Good
XXX	6 years	Meningitis	..			Normal	None	Poor	Poor
XXXI	Born deaf	..	..	Semi-deaf children.	G .... a^3 g^4 .... g^6 G .... a^6	Normal	Vowels and many monosyllables	Very good	Very good
XXXII	Born hearing	Scarlet fever at 2 years	..		G .... g^5 c^6 ... e^6 G .... a^1 a^3 .. c^4	R. Normal L. Scars	Vowels and some sentences	Very good	Very good
XXXIII	Probably born hearing	Probably Syphilis	..		c^1 .... a^4 c^1 .... a^4	R. and L. destroyed	Vowels and some sentences	Very good	Very good

Symptoms.—Apart from the deafness and the mutism, both of which have been referred to, the symptoms are not conspicuous. Tinnitus is not often complained of, but when it is inquired for many deaf mutes admit to have buzzing and other noises in the ears. These, however, do not seem to be distressing, and along with giddiness have probably passed away or become subdued after the earlier stages of the disease which has caused the deafness have passed away. Giddiness amongst deaf-mutes has been made the subject of careful investigation, and it seems that a larger proportion of deaf-mutes are free from sensations of giddiness when rapidly spun round than exists amongst hearing individuals; the explanation of this peculiarity is probably that in many deaf-mutes the semicircular canals are destroyed.

Diagnosis.—Except in children under a year old the diagnosis of deaf-mutism is not difficult. Shouting or bell-ringing behind the child produces no turning of the head and no alteration of the facial expression. Spoken words, unless when the child can see and therefore may read the lips of the speaker, are not reproduced. But during the first year of life, unless cases of deaf-mutism have already occurred in the family, there is no expectation or even suspicion of deafness. No suspicion may occur till it is discovered that speech is not developing as it usually does in children. It is therefore during the second year of life that the little patient is most often brought to the physician, and the parents, whilst alarmed at the non-appearance of speech, may adduce evidence of hearing which the physician cannot accept. Such evidence is the starting of the child when the door is slammed. This evidence may mean that a little hearing power exists, but most commonly means that the molecular disturbance or vibration has created an impression which the parents take to be the sense of hearing. The older the child, the easier does the diagnosis become, for a deaf child soon develops a language of signs and gestures to which a hearing child never needs to resort. Cases of mutism or dumbness without deafness sometimes come before the practitioner and form about 1 per cent of those applying for admission to the institutions for the education of the deaf and dumb. Most of these children are idiotic or at least weak-minded, but a few cases of true aphasia occur. Idiotic children are easily distinguished from the true deaf and dumb by the lack of animated facial expression, the smallness of the head, the narrowness of the arched palate, the lack of power to concentrate the attention, and so forth. Infantile aphasia, apart from weak-mindedness, is rare, and is probably due to disease of the speech centre in the brain. Deaf-mutism is occasionally simulated by adolescents or adults, but its diagnosis from true deaf-mutism is not difficult. In associating with the true deaf and dumb, the suspected person would almost certainly fail to use the gesture language which is acquired by most deaf-mutes, whilst under the influence of an anaesthetic he would betray himself when spoken to.

The otoscopic appearances in the ears of deaf-mutes are not characteristic. They are those—in acquired deafness—of the disease which has caused the deafness. Scarlet fever and measles cause loss of membrane, exfoliation of the ossicles, and necrosis of the walls of the tympanic cavity and the aditus ad antrum. Scars in the healed membrane are quite common. In congenital deafness the membrane is usually normal except in those cases—very common among deaf-mutes—in which post-nasal disease has caused Eustachian obstruction, rarefaction of the intra-tympanic air, and retraction of the membrane. But since the cause of deafness sufficient to cause dumbness is almost always in the internal ear, it follows that, with the exception of those cases in which active suppurative disease still persists, the otoscopic appearances in deaf-mute children are not of great clinical importance. The naso-pharynx in deaf-mutes is often filled with adenoid growths. In 30 per cent these are so marked that they should be removed both in the interests of the child's general health and with the object of improving his speech. The tonsils too are often enlarged, and in the orally taught impede proper speech.

The eyes in the deaf-mute are often affected; more than half the children in institutions fail when tested with Snellen's types even when the tests are not very rigidly applied. Examination with the ophthalmoscope shews in many cases changes which point strongly to inherited syphilis. Among 26 of the worst cases sent for ophthalmoscopic examination from the Glasgow Institution, there were two cases of hypermetropia, 4 of myopia, 14 of hypermetropic astigmatism, 5 of myopic astigmatism, and 1 of mixed astigmatism. The cornea was nebulous in 5 cases. In the retina degenerative changes were discovered in 9 cases, 7 with retinitis pigmentosa, of which 5 were incipient, 1 with choroido-retinitis, and 1 with round degenerative spots of discrete character.

The prognosis amongst deaf-mutes with regard to life and longevity is favourable; with regard to hearing very unfavourable. Cases of cure have been reported, but seldom on good authority. On the other hand, cases of improvement in, or development of, hearing occasionally occur. But even these occur so seldom that it is never wise to hold out any strong hope to the parents of a deaf and dumb child.

Treatment.—The study of the morbid anatomy of deaf-mutism, and the experience accumulating from a prolonged study of the deaf child, alike discourage us from expecting much from the treatment of that degree of deafness which results in dumbness, viz. surdism.

The prevention of marriage amongst the members of badly tainted fraternities, the prohibition of marriage between the congenitally deaf, and the prohibition of consanguineous marriages, would diminish the congenital deaf-mute rate. On the other hand, the prevention of the epidemic diseases which cause acquired deafness — meningitis, scarlet fever, and measles — would rapidly diminish the stream of acquired deaf-mutism. Both congenital and acquired deaf-mutism would lessen if the social conditions under which children are reared were improved. Deaf-mutism is very largely amongst the poor.

After surdism has taken place, treatment other than educational falls under two heads:—(1) The management of any middle-ear suppuration which may be present. This must proceed on ordinary surgical lines (*vide* p. 439). (2) The management of the nose and throat, which is so often obstructed in the deaf and dumb. This must consist of the removal of enlarged tonsils and of post-nasal adenoids, in the interests of the general health of the child, to facilitate the recovery of any middle-ear suppuration, and to make the articulation in the orally taught child more distinct.

Educational.—The first to educate deaf-mute children was the Spanish monk, Ponce de Leon. His method was oral—that is, he taught his pupils, who were the picked children of the wealthy families of Spain, to speak. Some 200 years later, namely about 1750, the French Abbé de l'Épée began the teaching of the average unpicked deaf-mute, and spent his time and his fortune in shewing that this could be successfully done. But de l'Épée substituted a system of signs for articulate speech. His chief critic was a German, Heinicke, who advocated articulate speech. Hence the contrasting names, French and German systems.

For 150 years the advocates of these methods have been at war, and there is not even now any indication that an understanding is likely to be reached. Teachers have regarded deaf children as a homogeneous class, and have applied one method to all children irrespective of the needs of the several groups into which deaf children should be divided. A scientific classification based on a clinical study of the deaf child affords a solution of the problem of methods.

In every country in the world deaf-mutes may be divided for educational purposes into these three groups:—(1) The semi-deaf and the semi-mute, consisting of 20 to 30 per cent of the whole. These children are for the most part intelligent, and their remnants of hearing and speech make them good pupils. Their voices are well intoned, and they are found in greater proportion in the higher than in the lower classes of a school. All the semi-mute and nearly all the semi-deaf have been born hearing. (2) Defective deaf-mutes. These compose about 15 per cent of the whole, and are of course the most backward in class. Amongst them are found a few semi-deaf and semi-mute children, cases of congenital syphilis with bad eyes, cases of meningitis in which not only hearing but intelligence has been damaged. But the bulk of them are true defectives—microcephalics, almost blind children, cases with the stigmata of degeneration well marked, and almost all congenitally deaf. They never advance far on any system of education, and most of them will never be self-supporting. (3) The average deaf-mute, comprising 60 per cent of the whole. The children here are of fair or bright intelligence. Any eye affections are chiefly errors of refraction, and may be corrected by the use of proper glasses. There are no mentally defective children amongst them, and their educational progress is such that at the end of the school period they are like group 1, quite fit to become self-supporting during adult life.

The objects of deaf-mute education are threefold—(1) To enable the deaf child to become self-supporting during his adult life; (2) to give the deaf child as much language as possible; (3) by the acquisition of speech to restore as many deaf-mutes as possible to communication with the hearing world. No method of education will make the defective deaf-mute self-supporting, and no amount of oral teaching will confer on him the ability to speak even fairly well. He should, therefore, be taught by the manual alphabet method.

On the other hand, either method of education will enable the average deaf-mute and the semi-deaf and semi-mute child to use language properly.

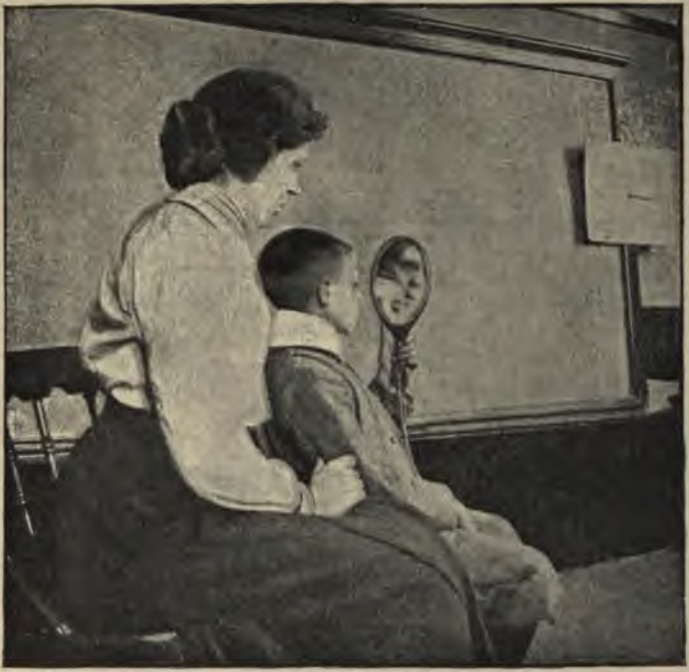
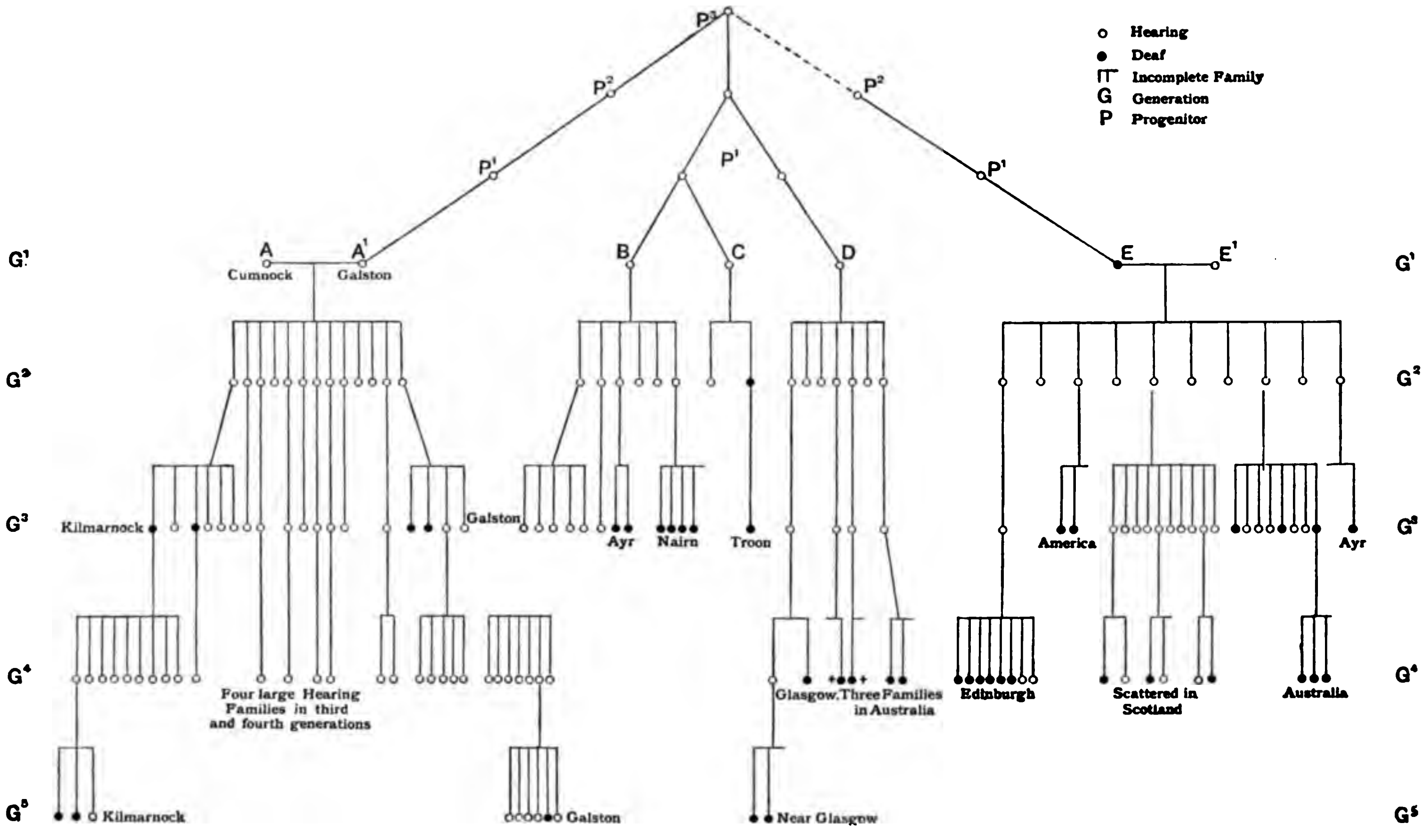


FIG. 59.—Oro-acoustic method of teaching. The teacher speaks into the ear of the pupil, who also sees her lip movements in the mirror. (After Bezold.)

The manually taught child will at the end of his school life be unable to communicate (except in writing) with the hearing part of the population, whereas the orally taught child will, even if his speech and lip-reading be imperfect, as it almost always is, be able to communicate by speech with those hearing people with whom he comes into daily contact. He has all that the manually taught child has, and also this power of speech and lip-reading—a power and advantage great enough to make the oral method the one to be chosen except in cases in which failure is likely to result.

A careful clinical study of the deaf child renders it possible to make a classification that will help the teacher to work effectively, and shew him how to avoid placing the child under the wrong method, and thus



THE AYRSHIRE FAMILY OF DEAF-MUTES.

A, possibly related to A^1 (his wife); A, second cousin to B, C, and D; D, cousin to brothers B and C; E, related to A, B, C, and D. The tree shews 41 deaf-mutes. See *Deaf-Mutism*, by J. K. Love and W. H. Addison. (Glasgow: James MacLehose and Sons. New York: The Macmillan Co., 1896.)

In the above tree A married A^1 of the same name but not known to be related before marriage to A. B, C, and D are all second cousins to A. E was born in New York, and absolute proof of his relationship to A has been got, though the exact relationship has not been made out. E returned to Scotland in early life. Since 1896 (when this tree was constructed) several deaf children have been born into the family in or near Glasgow, and have been admitted to the Glasgow Institution for the Education of the Deaf and Dumb. The family tree has been carefully traced from the marriage of A about the end of the eighteenth century. The progenitors before that date are hypothetical.

injuring him. Further, such a classification will bring the war of methods to an end.

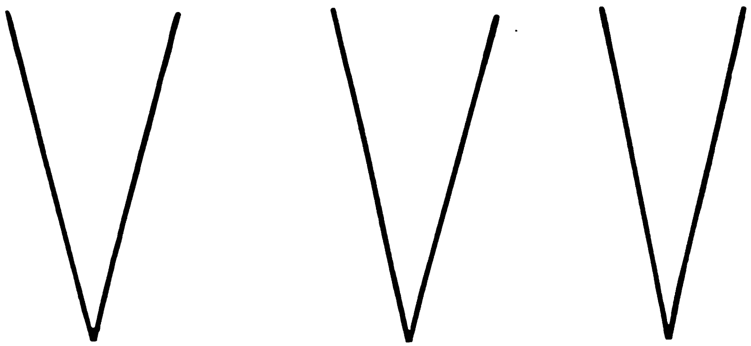
The examination of the ears and the testing of the hearing of the children of elementary schools shew that a large number have defective hearing for such tests as the ticking of a watch and the stroke of an acoumeter, and even for whispered speech, but the percentage whose deafness is so great that they cannot follow easily the ordinary class work is probably not more than 2 per cent, and may be nearer 1 per cent. But this percentage means that there are ten times as many children hard of hearing as there are of the deaf and dumb, and that in London alone there are probably 5000 to 10,000 children in the elementary schools whose backwardness is due to hardness of hearing.

The following is a scheme for dealing with the educational treatment of all children suffering from mere hardness of hearing to total loss of hearing, the whole clinical picture of the deaf child having been considered :—

SCHEME OF EDUCATION FOR CLASSIFIED DEAF

25 Per Cent. 60 Per Cent. 15 Per Cent.

Hard-of-Hearing Children. Semi-Deaf Better Cases. Semi-Deaf Worse Cases. Average Deaf-Mute Oral Successes. Oral Failures. Defective Deaf Mutes.



Special Classes in Hearing Schools. Special Day School or Oral Residential School. Separate Classes of Manual Alphabet School (Residential).

The logical outcome of the adoption of this scheme would be the extinction of the Institutional system for all but the defective deaf and a small number of the average deaf-mutes. But the poverty and the bad social environment of the deaf child in our largest cities make the realisation of this ideal attainable only by slow degrees.

James Kerr Love.

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J. K. L.

AIDS TO HEARING

By JAMES KERR LOVE, M.D.

IN deafness of high degree, ordinary conversation, and even loud speech, cannot be distinguished by the unaided ear, and some instrument fitted to collect, increase, or concentrate the voice sounds must be employed.

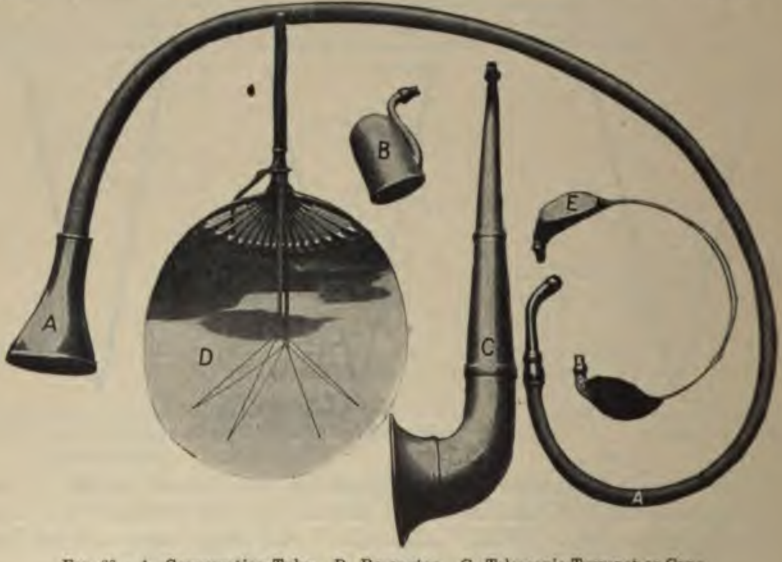

FIG. 60.—A, Conversation Tube. B, Resonator. C, Telescopic Trumpet or Cone. D, Audiphone or Auditory Fan. E, Double Auricle with Retaining Head-band.

These are called "aids to hearing." The best aid to hearing is good eyesight trained to lip-read, or rather to speech-read the speaker. Most very deaf persons with good eyesight can be trained to lip-read those with whom they come in daily contact.

The instruments meant to aid the hearing power of the very deaf shew much variety of construction, and are based on no single principle of acoustics. There is no comparison between the spectacles fitted for defective sight and the instruments used for defective hearing, because although sound is refracted and reflected like light, the media for accomplishing this for sound are much clumsier and less portable than a pair of spectacles. When a deaf person cannot hear the raised voice at a distance of half a yard, some form of hearing-trumpet or conversation-tube should be used. For ordinary conversation a tube made of spiral wire and covered with woven silk or cotton is the best. Such a tube has an ear-piece to fit the meatus of the deaf person, and widens in its length of about a yard to a bell-shaped vulcanite cup into which the person addressing the deaf person speaks. The dispersion of sounds according to the law of inverse squares is prevented, and the concentrated but not too loud sound is conveyed to the ear. Such a tube, which can be rolled up and put into the pocket, is useless in a church or public hall; it can only be used by one speaker at a time, and its use directs the attention of all to the infirmity of the deaf person.

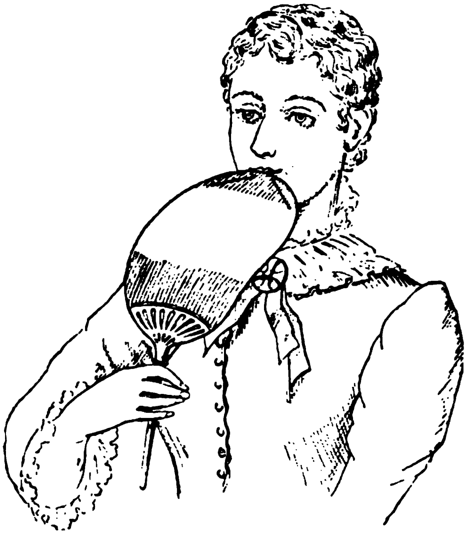

FIG. 61.—The Audiphone or Auditory Fan in use.

Cones and trumpets held by the hand to the ear make good collectors of sound. Generally the large forms are better than the small, because the amount of sound gathered is greater. Bell-shaped resonators are much used as "aids" by deaf people because they are small and easily carried in the pocket, and because they lend themselves better to the ornamental forms. To all the instruments above mentioned two objections are raised: they are conspicuous and they engage one hand of the deaf person. Instruments fastened about the head are much in request, and are called "auricles." In the case of women these may be of considerable size, and may be concealed under the hair. In men a spring passes over or behind the head and keeps the "auricle" applied to the side of the head. Many of these are powerful instruments, and may be applied to one or both ears.

In some cases in which bone conduction is well preserved the "audiphone" or auditory fan makes an elegant and effective "aid," and is specially suitable for women. It consists of a fan-shaped sheet of vulcanite, the convex border of which is held between the incisor teeth while the handle is held in the hand. By a series of diverging cords a curvature may be given to the instrument which increases its efficacy. For table use large receivers may be used, the instrument lying on a table, while flexible conductors, after the principle of the binaural stethoscope, are led to each ear of the wearer.

Within recent years the micro-telephone has been much exploited as an "aid." It is well known that many deaf persons hear the telephone quite well. The instrument consists of a receiver which is fixed to the vest or dress front, a small battery of dry cells which is carried in the pocket, and a telephone ear-piece which is either held to the ear by the hand or fixed against the ear by means of a spring passing round or over the head. Cords connect the battery with the receiver and the ear-piece. The receiver may be enlarged or duplicated, and the cords lengthened for use in church or lecture hall. Although this arrangement suits a small percentage of cases better than the ordinary collectors and resonators already referred to, it is not useful in such a large number of cases.

Speaking generally, nothing but experiment will determine the particular "aid" which will best suit a deaf person. Perhaps this uncertainty is chiefly because the same degree of deafness or hardness of hearing may arise from quite different causes, for example, otosclerosis and aural catarrh.

In the education of the deaf and dumb, aids to hearing have been used to a limited extent. The cases suitable are those of semi-deaf children. The form of instrument used is a double conversation-tube, one of the speaking ends of which is used by the teacher, the other by the deaf child, whilst the single hearing end is held in the external auditory canal of the child. I have visited most of the best schools for the deaf on both sides of the Atlantic, and have not found teachers generally of the opinion that either these conversation-tubes or the micro-telephone instruments which have been more recently proposed as their substitutes are of much practical value. It is urged that they take the attention of the child from lip-reading, which even in the case of the semi-deaf must be the great "aid" to what hearing is left. Except in the form of advertisement there is not much written on this subject, but a good article occurs in Politzer's *Textbook on Diseases of the Ear* (Baillière, Tindall, and Cox, London, 1902), and the *Catalogue of Otacoustical Instruments*, by Mr. Hawksley of 357 Oxford Street, London, is very useful.

JAMES KERR LOVE.

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